ARCHIVES OF PEDIATRICS

May 1960

New York Medical College, Flower and Fifth Avenue Hospitals

"Examine your armamentarium! It's not complete without 'BABY SILICARE' for diaper dermatitis"

Medicated Baby Silicare Powder and Lotion can help you in the management of even the most difficult cases of diaper dermatitis. Superior clinical effectiveness of both Powder and Lotion is well documented in the literature.[1,2,3] They are routine on obstetric and pediatric services of many leading hospitals. Patient acceptance is high. Why not use Baby Silicare Powder and Lotion for prevention and treatment of diaper dermatitis?

1. Kaessler, H. W.: Arch. Ped. 74:47 (Feb.) 1957.
2. Kahan, H. *et al.*: Arch. Ped. 73:125 (Apr.) 1956.
3. Editorial: J.A.M.A. 165:254 (Sept. 21) 1957.

MEDICATED

'Baby Silicare'®

POWDER AND LOTION

active ingredients:
glyoxyl diureide
dimethylpolysiloxane
hexachlorophene

REVLON PHARMACAL DIVISION • NEW YORK, N. Y.

TO STOP DIARRHEA

from all points... growing evidence favors

FUROXONE®

brand of furazolidone

■ Pleasant-flavored LIQUID, 50 mg. per 15 cc. (with kaolin and pectin) ■ Convenient TABLETS, 100 mg. ■ Dosage—400 mg. daily for adults, 5 mg./Kg. daily for children (in 4 divided doses).

SWIFT RELIEF OF SYMPTOMS

EFFECTIVE CONTROL OF "PROBLEM" PATHOGENS
(no significant resistance develops to this wide-range bactericide)

WELL TOLERATED, VIRTUALLY NONTOXIC

NORMAL BALANCE OF INTESTINAL FLORA PRESERVED
(no monilial or staphylococcal overgrowth)

From a Large Midwestern University:

FUROXONE CONTROLS ANTIBIOTIC-RESISTANT OUTBREAK

An outbreak of bacillary dysentery due to Shigella sonnei was successfully controlled with FUROXONE after a broad-spectrum antibiotic had proved inadequate. Cure rates (verified by stool culture) were 87% with FUROXONE, 36% with chloramphenicol. Only FUROXONE "failures" were those lost to follow-up. Chloramphenicol failures subsequently treated with FUROXONE responded without exception. FUROXONE was also used effectively as prophylaxis and to eliminate the carrier state. It was "extremely well tolerated in all 191 individuals who received it either prophylactically or therapeutically." Galeota, W. R., and Moranville., B. A.: Student Medicine (in press)

EATON LABORATORIES, NORWICH, NEW YORK

ARCHIVES OF PEDIATRICS

ALBRO C. GAYLOR, *Publisher*
ROSE E. BODET, *Assoc. Publisher*

JOHN FITCH LANDON, *M.D., Editor*

Vol. 77 No. 5

May 1960

CONTENTS

Published monthly since 1884.
Subscriptions: one year—$7.50; two years—$12.00; three years—$16.00.
Single Copy—$1.00

Back issues older than two years are available through Walter J. Johnson, Inc., 111 Fifth Avenue, New York 3, New York.

CLEARS SCALP SEBORRHEAS
CRADLE CAP TO DANDRUFF

Sebical Cream has been found to be extremely effective in the treatment of most seborrheas of the scalp. It offers in a single preparation, these clinical advantages:

relieves itching promptly / removes scales and crusts, clears scalp oiliness / reduces inflammation / provides antimicrobial action /

NEW

In a recent clinical study* of infants and children with severe seborrhea capitis (cradle cap), all cleared completely on Sebical. Duration of therapy was 7 to 28 days. Sebical is easy to apply, nonstaining, virtually nonirritating, nontoxic, nonsensitizing.

FORMULA: Allantoin 2%, hexachlorophene 1%, special Coal Tar Extract (Tarbonis R) 2%, in a special penetrating, nonstaining, vanishing base.

APPLICATION: Rub Cream into scalp one to three times daily, depending on severity of condition. Cleanse scalp with Sebical Shampoo.

SUPPLIED: Tubes of 2 oz.

*Karel, J. R. and Najmabadi, A.: Archives of Pediatrics 77: 94-98, 1960.

SEBICAL™
CREAM

FROM CRADLE CAP TO DANDRUFF

REED & CARNRICK • KENILWORTH, N. J.

New York Medical College

Commemorates

One Hundredth Anniversary

Nucleus of the city's newest medical center, the New York Medical College faculty has grown from an original 8 to more than 800 physicians—the student body from an original 59 to 490. In addition to staffing the college's voluntary hospitals, Flower and Fifth Avenue is responsible for the medical programs of such municipal institutions as the new Metropolitan and Bird S. Coler Hospitals.

Within the next two years, a new cardio-pulmonary institute and residence facilities for students and nurses are planned. Today with its affiliated hospitals, the college facilities provide more than 3000 beds for patient care, teaching and research, and medical care for over a million patients annually.

The Cover . . .

This artist's drawing shows more completely the scope of college and hospital units. Originally located at East 20th Street near Third Avenue, Flower Hospital moved to its present location at Fifth Avenue and 106th Street in 1935, sharing a building with Fifth Avenue Hospital. In 1938, a merger gave the institution its present corporate title . . . "New York Medical College, Flower and Fifth Avenue Hospitals".

a new film

"Soma in Cerebral Palsy"

by Catherine E. Spears, M.D.

WALLACE LABORATORIES is pleased to announce the availability of the 16 mm., color film, SOMA IN CEREBRAL PALSY.

Photographed at the Children's Country Home in Westfield, New Jersey, and narrated by Dr. Spears, this 14-minute sound film presents the results of the preliminary clinical trials with the new muscle relaxant, SOMA[T.M.] (carisoprodol Wallace).

Four children were selected for this presentation. They are representative not only of the type and degree of handicap seen at this center, but also of the progress attained with the help of SOMA.

Copies of the film for showing to medical groups may be obtained by writing to the Professional Services Department, Wallace Laboratories, New Brunswick, New Jersey.

WALLACE LABORATORIES
NEW BRUNSWICK, NEW JERSEY

General Information . . .

Archives of Pediatrics is an independently-owned monthly magazine for pediatric specialists and other doctors whose original material and research data has significance for this journal's important reading audience.

Contributions are invited from practicing physicians and clinicians whose ancillary services include such specialties as pathology, radiology, odontology, psychology, etc. Only manuscripts not previously published will be considered. Views and statements expressed are the sole responsibility of the author. Please send original and one copy, double-spaced, typewritten, to the attention of the Editor at address below.

Illustrations—number clearly, include legends and author's name. (X-rays do not reproduce well.) A reasonable number of halftones will be reproduced without charge to the author; lengthy charts, tables and extra illustrations must be handled by special arrangement.

Copyright protects all original material. Publisher's permission must be requested for any and all reproduction.

Reprints of articles must be ordered promptly after receipt of galleys. Prices will be sent on request. (Minimum 250)

Subscription Rates: U. S. and Possessions, $7.50 per year (students, interns and residents, $4). Canada, $7.75 . . . Foreign, $8.25. Two years, $12. Three years $16. Single copy price $1.00. Please send check, draft, postoffice or express money order, and give six weeks' notice of change of address, name of subscriber's old, new address, and zone number.

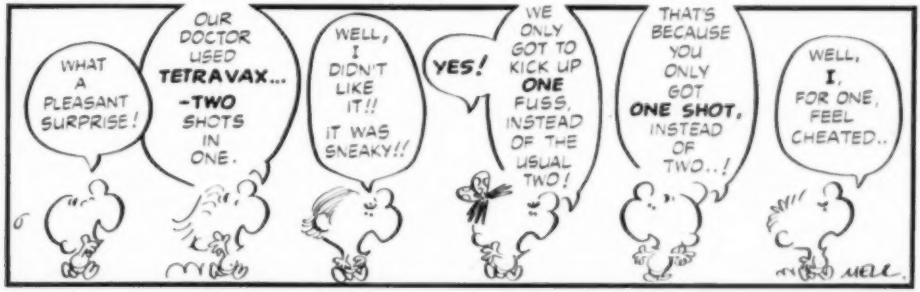
ARCHIVES OF PEDIATRICS

200 Park Avenue South at 17th St., New York 3, N. Y.

FOR SIMULTANEOUS IMMUNIZATION AGAINST 4 DISEASES:

Poliomyelitis-Diphtheria-Pertussis-Tetanus

PEDI-ANTICS



TETRAVAX®

DIPHTHERIA AND TETANUS TOXOIDS WITH PERTUSSIS AND POLIOMYELITIS VACCINES

now you can immunize against more *diseases... with* fewer *injections*

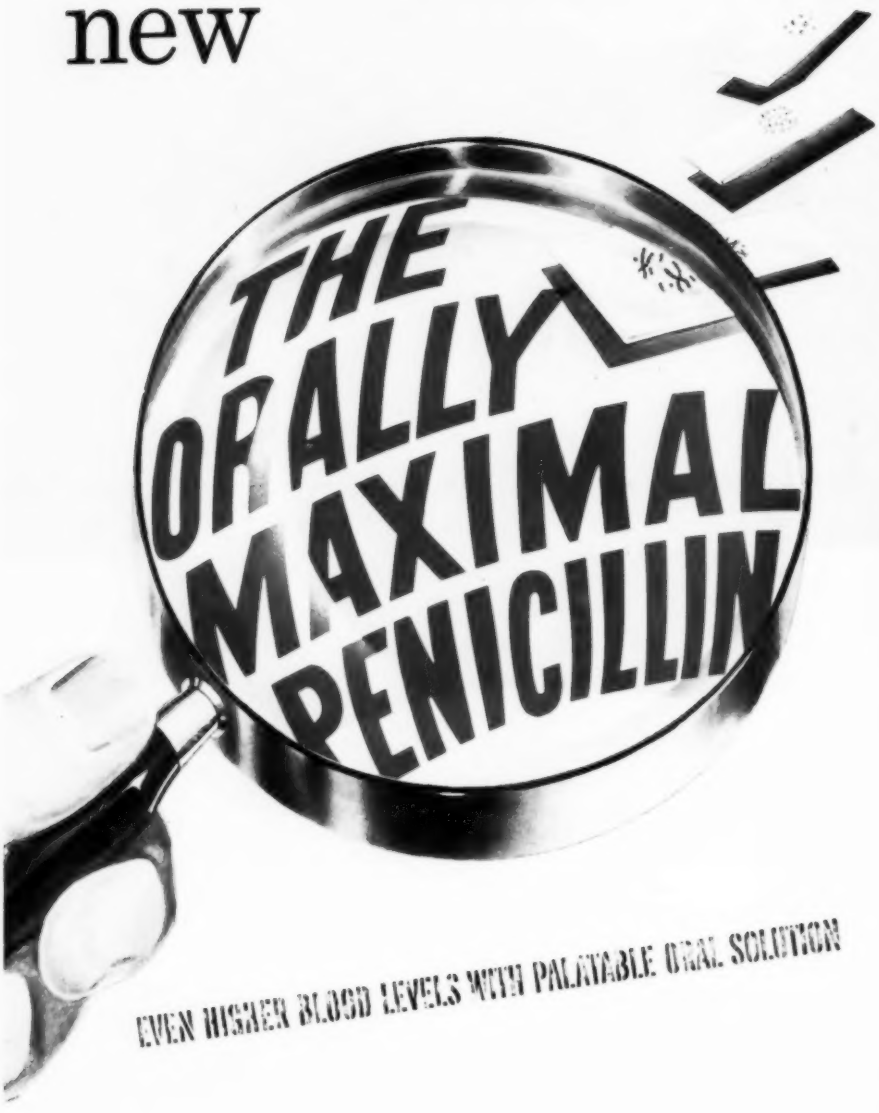
Dose: 1 cc.
Supplied: 9 cc. vials in *clear plastic* cartons. Package circular and material in vial can be examined without damaging carton. Expiration date is on vial for checking even if carton is discarded.

For additional information, write Professional Services, Merck Sharp & Dohme, West Point, Pa.

TETRAVAX IS A TRADEMARK OF MERCK & CO., INC.

MERCK SHARP & DOHME, DIVISION OF MERCK & CO., INC., WEST POINT, PA.

new



EVEN HIGHER BLOOD LEVELS WITH PALATABLE ORAL SOLUTION

MAXIPEN

α-phenoxyethyl penicillin potassium
(phenethicillin potassium)

THE ORALLY MAXIMAL PENICILLIN

MAXIMAL ABSORPTION
Acid stable. Highly soluble.

MAXIMAL BLOOD LEVELS
Higher than oral potassium penicillin V; higher than intramuscular procaine penicillin G.

MAXIMAL ORAL INDICATIONS
Infections caused by streptococci, pneumococci, susceptible staphylococci, and gonococci.

MAXIMAL FLEXIBILITY
May be administered without regard to meals. However, highest absorption is achieved when taken just before or between meals.

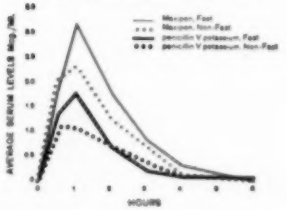
A Triumph of Man Over Molecule...
a product of Pfizer Research

Dosage: For moderately severe conditions, 125 to 250 mg. three times daily. For more severe conditions, 500 mg. as often as every 4 hours around the clock.

Note: To date, MAXIPEN has not shown less allergic reactions than older oral penicillins. Usual precautions regarding penicillin administration should be observed.

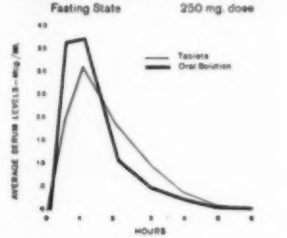
Supplied: MAXIPEN FOR ORAL SOLUTION; reconstituted each 5 cc. contains 125 mg., in 60 cc. bottles. Also 125 mg. and 250 mg. tablets.

COMPARATIVE ORAL SERUM LEVELS*
Fasting and Non-Fasting States / 250 Mg. Dose



*Based on 3294 individual serum antibiotic determinations. Complete details on request.

COMPARATIVE SERUM LEVELS
MAXIPEN TABLETS VS. ORAL SOLUTION
Fasting State 250 mg. dose



New York 17, N.Y.
Division, Chas. Pfizer & Co., Inc.
Science for the world's well-being™

Why Clinical Judgment Often Dictates Altafur for Peroral, Systemic Therapy of Pyodermas

Gratifying Therapeutic Response

ALTAFUR was found "highly satisfactory in most of the primary and secondary bacterial dermatoses treated to date," including "pyodermas . . . caused by antibiotic resistant strains of staphylococci."[1] In a nationwide survey[2] there were **94%** satisfactory results (cured or improved) among 159 patients treated with ALTAFUR for pyodermas.

Virtually Uniform in vitro Susceptibility of Staphylococcus aureus

99.5% of isolates (214 of 215) from patients with staphylococcal infections—including many antibiotic-resistant strains—proved sensitive in vitro to ALTAFUR in tests conducted across the nation.[3] **99.7%** of staphylococcal isolates (334 of 335) at a large general hospital—including many antibiotic-resistant strains—proved sensitive in vitro to ALTAFUR.[4]

Wide, Stable Antimicrobial Spectrum

"Because of its relationship to previously developed nitrofurans, it is anticipated that [ALTAFUR] will retain its original spectrum after longstanding clinical usage."[5] Development of significant bacterial resistance to ALTAFUR has not been encountered to date.[6]

Minimal Side Effects

Side effects are easily avoided or minimized by these simple precautions: 1) *alcohol should not be ingested in any form, medicinal or beverage, during* ALTAFUR *therapy and for one week thereafter* 2) each dose should be taken with or just after meals, and with food or milk at bedtime (to reduce the likelihood of occasional nausea and emesis).

---

1. Weiner, A. L.: Paper presented at the Conference on Recent Advances in the Treatment of Chronic Dermatoses, University of Cincinnati (Ohio), Nov. 5, 1959. 2. Compiled by the Medical Department, Eaton Laboratories, from case histories received. 3. Christenson, P. J., and Tracy, C. H.: Current Therapeutic Research 2:22, 1960. 4. Glas, W. W., and Britt, E. M.: Proceedings of the Detroit Symposium on Antibacterial Therapy, Michigan and Wayne County Academies of General Practice, Detroit, Sept. 1, 1959, p. 14. 5. Leming, B. H., Jr.: Ibid., p. 22. 6. Investigators' reports to the Medical Department, Eaton Laboratories.

Tablets of 250 mg. (adult)
and 50 mg. (pediatric)
bottles of 20 and 100

Altafur®

NITROFURANS . . . a unique class of antimicrobials
EATON LABORATORIES, NORWICH, NEW YORK

Richard E. Smith
121 North Main St
Furunculosis, severe

32
City

NAME Richard E Smith AGE 32
ADDRESS 121 North main St DATE 3/25/60

℞

Tab Altafur 250 mg
Disp no XX
Sig: 1 tab qid c̄ food or milk

R.B.

baby the
infant's skin
with

PANTHODERM® CREAM

**relieves itch and pain • promotes healing •
guards against irritation and chafing**

in 2 oz. and 1 lb. jars;
1 oz. tubes.

IN

DIAPER RASH
EXCORIATED BUTTOCKS
CHAFING, HEAT RASH
INTERTRIGO
ITCHING

Remarkably effective . . . often when other therapy fails . . . Panthoderm Cream treats the infant's skin with "tender, loving care." Clinically it has shown evidence of "epithelizing stimulation . . . an antipruritic effect . . . an antibacterial effect . . . in a variety of dermatoses" such as external ulcers, burns, wounds, pruritus vulvae, a variety of dermatoses. Minimum risk of sensitization.

Dainty as a fine cosmetic, Panthoderm Cream is clean, snow-white, non-staining, water-miscible.

Samples yours for the asking.

u. s. vitamin corporation • PHARMACEUTICALS
Arlington-Funk Laboratories, division • 250 East 43rd Street, New York 17, N. Y.

NEW CLINICAL EVIDENCE

offers additional proof that

CREAM of RICE

IS EASIER TO DIGEST

than any other kind of cereal!

In a study conducted with intubated men and women, each received a cup of Cream of Rice, oatmeal and farina on different days. From each subject samples were withdrawn periodically and tested for sugar. The amount of sugar tested was taken as the index of digestibility.

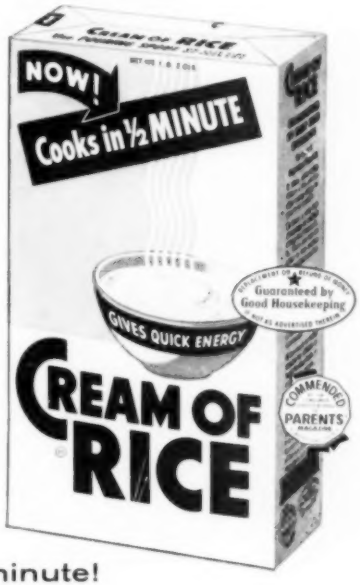
Results showed that Cream of Rice was easier to digest and gave quicker food energy than the other cereals tested.

RECOMMENDED BY LEADING PEDIATRICIANS

More and more pediatricians are recommending Cream of Rice as one of baby's first solid foods. Pediatricians are also recommending it for growing children—because it's so rich in food value.

Both adults and children love the deliciously different taste of Cream of Rice. That's why it's so good as a breakfast cereal, too!

Cream of Rice is non-allergenic, low in sodium, low in fat—rich in Vitamin B_1, Riboflavin, Niacin and Iron.



Cooks in ½ minute!

WRITE FOR PROFESSIONAL SAMPLES TO:
GROCERY STORE PRODUCTS CO.
Dept. C5A, West Chester, Pa.

PERKINS, F. T., YETTS, R., AND GAISFORD, W.: A Comparison of the Responses of 100 Infants to Primary Poliomyelitis Immunization with Two and with Three Doses of Vaccine. (British Medical Journal 5129:1083 April 25, 1959).

The response of infants to poliomyelitis vaccine is governed by two factors—the level of maternal antibody and the number of antigenic stimuli given in the course of primary immunization. Two groups of infants, 45 aged 1 week and 55 aged 16 weeks, were studied. The 1-week-old infants had such high levels of maternal antibody that doubling the volume and giving three doses of vaccine resulted in no better response than that given by the normal immunization schedule in similar infants in the previous study. Satisfactory responses were obtained with the ordinary dose of 1 ml. in the 16-weeks-old infants who had very low maternal antibody levels, especially after the third dose of vaccine. In this age group, however, inhibitory levels of maternal antibody were present in some infants, and in order to obtain satisfactory immunization to all types in all infants it is suggested that immunization should be delayed until 6 to 9 months of age, at which time three doses of vaccine should be considered as a course of primary immunization.

Author's Summary

OUTSTANDING

to prevent
and clear up

diaper rash

DESITIN®
OINTMENT

physically Desitin Ointment assures constant protection against the irritation of urine and excrement.

bacteriostatically it markedly inhibits ammonia-producing bacteria.

therapeutically Desitin Ointment soothes, lubricates—and stimulates healing by means of high grade cod liver oil, rich in vitamins A and D and unsaturated fatty acids.

samples *and literature available from...*

DESITIN CHEMICAL COMPANY • 812 Branch Avenue, Providence 4, R. I.

Dependable Vitamin "Blocks"

BUILD SOUND BODIES DURING CRITICAL FORMATIVE YEARS

Pluravit® Drops

A pleasant lime flavored, readily dispersible combination of 8 essential vitamins

Each 0.6 cc. contains:

Vitamin A	5000 units
Vitamin D_2	1000 units
Vitamin B_1	1 mg.
Vitamin B_2	0.4 mg.
Vitamin B_6	1 mg.
Nicotinamide	5 mg.
Pantothenic acid	2 mg.
Vitamin C	50 mg.

Stable • Mixes Readily

DOSAGE: Infants 0.3 cc., children and adults 0.6 cc. daily.
May be taken directly or stirred in milk, fruit juices, or other food.

How supplied: Bottles of 15 and 50 cc. with graduated dropper.

Winthrop LABORATORIES
NEW YORK 18, N. Y.

Pluravit, trademark reg. U. S. Pat. Off.

guest editorials

Let There Be No Polio This Year

HAROLD JACOBZINER, M.D., *Assistant Commissioner, Maternal and Child Health, The City of New York, Department of Health*

Five years ago this month, Dr. Thomas Francis, Jr. announced to the medical profession that the antipolio vaccine (Salk) is *safe* and effective. This statement was based on extensive field trials meticulously conducted and critically analyzed. With each passing year the original statement gains in accuracy. We have failed, however, to capitalize fully on its potential significance by not applying more widely the available knowledge and tools at our disposal.

MAGNITUDE OF THE PROBLEM

Last year the number of cases of poliomyelitis in the United States was much higher than reported during the two preceding years as evidenced by the following figures.

	1955	*1956*	*1957*	*1958*	*1959*
Paralytic	10,063	6,717	2,164	3,113	5,694
Total	29,298	15,465	5,949	6,033	8,577

The number of paralytic cases in 1959 almost approached the 1956 level. Nearly 67% of all reported cases were of the paralytic type, a much higher percentage than in the two preceding years. The greatest concentration of paralytic cases was in the preschool group. Forty-three percent of all paralytic cases occurred in children under 5 years of age with type one polio virus predominating. Five to nine year olds were more fortunate, having lower attack rates due to a higher vaccination status since they were the lucky recipients of the vaccine through the NFIP school vaccination program.

CHARACTERISTICS

There were 3 major epidemics in 1959 in urban areas with attack rates of over 15/100,000 of the population and numerous widely scattered localized concentrations and a number of moderate urban epidemics with attack rates ranging from 5 to 14.9/100,000 and a

number of rural outbreaks. The 3 major epidemics occurred in Des Moines, Iowa, Kansas City, Missouri and Little Rock, Arkansas. In all of these epidemics in the highest concentration was among the lower socio-economic groups in crowded and depressed neighborhoods and with unusually high attack rates among the negroes and the unvaccinated. This pattern has been repeatedly observed since 1956.

RELATION OF ATTACK RATE TO VACCINATION

Eighty two percent of the total paralytic cases reported occurred among the unvaccinated or partially vaccinated. Over 60% of the paralytic cases in the United States did *not* have *any* Salk vaccine and over 18% of the paralytic cases had only one or two doses. Only 12% of the paralytic cases had 3 doses and 3% of all paralytic cases had four injections. Immunization surveys conducted in various parts of the country disclosed that many areas contained poorly immunized populations and that the highest immunization rate in the lower socio-economic population groups was nearly always lower than the lowest rates reported for the upper socio-economic groups. The relation between vaccination status and attack rate is thus clearly apparent. There is a distinct inverse ratio between level of immunization and incidence of paralytic polio. The highest incidence occurred in poorer sections where the percentage of immunized persons is low.

During 1959, the same experience was obtained in New York City. Seven low socio-economic districts out of a total of 30 contributed 75, out of a total of 168, over 45% of all reported cases of poliomyelitis. Fifteen percent of all cases occurred in *one* district (Central Harlem) inhabited chiefly by non-whites. The attack rate in the Negro was much higher than in the general population accounting for nearly 50% of all cases, though they constitute less than 20% of the total population. Eighty-two percent of all cases were of the paralytic type, a 32% increase over 1958. Fifty percent of all reported cases occurred in children under 5 years. Prior to 1955, the 5-9 year age group had the highest attack rate. The shift to the preschool group is undoubtedly related to the level of immunization.

Of the 20 fatal cases, 11 or 55% received *no* vaccine at all. Only 4 of these received 3 injections and none of the individuals who died received four injections. It may also be mentioned that poliomyelitis is appreciably more severe in the adult. The 25 year age

group and over, while accounting for 19 out of the 168 cases, contributed 7 of 20 deaths, a case fatality rate of 35%. Six of the seven fatal cases in this adult group had no vaccine at all and only one had 3 doses.

An analysis of the paralytic cases in the under 5 year age group in 1958 in New York City indicated that the specific case rates were 3.9/100,000 of the population for the White, 8.7 for the Negro and 18.0 for Puerto Rican children. Thus the paralytic attack rates were much higher in the Puerto Rican and the Negro than in the White. A survey relating to immunization status likewise indicated that the percentage of immunizations was lower in the Negro than in the White and the lowest percentage was observed among the Puerto Rican, possibly due to a lack of communication.

In several studies in the United States by social scientists it was also found that the uneducated and the poor have the poorest level of protection and that the routine audio-visual aids are not effecive ways of reaching these groups. More intensive personal efforts are needed.

While on the negative side it must be admitted that among children under 5 years about 4½ million as yet received no vaccine at all, notable progress in morbidity and mortality has been achieved. In New York City, for example, the median number of annual cases for the years 1947-1956 was 651. In 1959, however, admittedly a peak year, 168 cases were reported of which 143 were paralytic. In 1957, apparently a low year, we had only a total of 57 cases of which 39 were paralytic. It is obvious that in order to eradicate poliomyelitis a high level of immunization among preschool children will be required—from 90-95%.

IS THE VACCINE SAFE AND EFFECTIVE

Over 300 million doses of vaccine have been used without evidence of any break in safety. The effectiveness of the vaccine has actually been underestimated by Dr. Francis in his original monumental report.

During the last two years a level of over 90% protection has been obtained for children under 15 years of age who received three or more doses. Its effectiveness in combating paralytic polio is thus unquestioned. To obtain a satisfactory immunization level, however, a basic series of 3 doses is required and a 4th injection not later than one year following the third dose. Booster injections

should also be administered to preschool children at 3 years of age and prior to entrance to school. The quadruple antigen (DPTP) may conveniently be used for immunizing preschool children.

WHAT MUST BE DONE?—A PROGRAM OF ACTION

The practicing physicians and particularly pediatricians have written a brilliant epic in medicine by the control of and virtual elimination of certain communicable diseases of childhood through widespread immunization efforts. There is no reason why they should be less successful in the control of poliomyelitis. The vulnerable groups have been identified. Every physician must make a determined and vigorous effort to completely immunize all preschool children and their families as promptly as possible, prior to the onset of the polio season.

We must assume our responsibilities as pediatricians and physicians in accordance with the following unanimous recommendation made by the House of Delegates of the American Medical Association on March 4, 1958.

"1. Each physician assumes the responsibility for making certain whenever possible that all members of families he serves receive protection against poliomyelitis by having the full three doses of polio vaccine;

2. State medical organizations arrange with state health departments for a joint effort to bring together county medical society representatives and representatives of county and city health departments for the purpose of discussing the need for joint study committees at the local level to survey the problems which may exist and to work jointly to solve them;

3. County medical societies meet with county and local health department representatives to create study committees to survey the problem of immunization as it may exist in the local area and develop and implement a satisfactory program to meet the local situation".

In this year, 1960, the Golden Anniversary of the White House Conference on Children and Youth is being called. Its purpose is "to promote opportunities for children and youth to realize their full potential for a creative life in freedom and dignity." We as physicians can help greatly in accomplishing this objective and in giving meaning to these words by eliminating poliomyelitis and by protecting every child regardless of race, color or creed or socioeconomic status from the ravages of this disease by immunizing all preschool children in our private and clinic practice.

Emotional Disturbances in the Neo-Natal Period and Early Infancy

REGINA M. FITTI, M.D.* AND J. M. VAN HAUVAERT, M.D.**
Pennsylvania

RECENT literature has contained many articles on emotional disturbances in children, but few of these articles discuss emotional disturbances or possible emotional disturbances in the neo-natal period or early infancy. Such disturbances may be more common or more readily recognized in institutions, where the infant has been separated from the mother at birth or in early infancy; for socio-economic or other reasons, than in practice.

We have been impressed by the apparently large number of infants from the age of 5 days, who presented problems, for which we were unable to account on a physical basis or to demonstrate laboratory abnormalities. Most of these infants fitted into one of two fairly well defined patterns of behavior. One type we have designated as the apathetic infant, the other as the hyper-active or anxious infant.

The apathetic infant seems to exist in a prolonged intra-uterine state, where his requirements have no bearing on his reactions. He seldom moves, spends most of the time curled in the fetal position. He appears withdrawn, completely disinterested. Physical examination is within normal limits, except that the reflexes, sucking, grasping. Moro and patellar are sluggish or absent. He is very pale and white. He seldom cries and when he does, it is a few half-hearted wails. He does not sleep all the time, merely lays curled in the bassinet, looking depressed, unloved and unwanted. He eats poorly, does not cry when hungry, and gives up sucking after a few pulls. He shows an apparently poor stress reaction. Without supportive therapy to maintain basic nutritional requirements, a rapid

* Medical Director, St. Vincent's Hospital for Women & Children, Philadelphia, Pennsylvania.
** Resident in Pediatrics, Hahnemann Hospital, Philadelphia, Pennsylvania.

downhill course, marked by grayish pallor and cold clammy skin, suggestive of shock, occurs.

Gesell and Ilg have described infants who rarely give evidence of hunger by crying or show little or no eagerness for food. They feel this may be due to inborn poor appetite. This anorexia may persist for months, but the child seems to progress in other respects.

After responding to treatment, the apathetic infants we have observed, take their feedings with the eagerness and gusto seen in most infants. These infants do not fit exactly the picture presented by Gesell and Ilg.

The hyperactive infant is jittery, with generalized hyperactive reflexes. He moves constantly, squirming about in the bassinet; sleeps short periods. The knees, toes and face may be raw from rubbing against the bed linen. The swallowing reflex is frantic and ineffective, allowing the infant to swallow more air than formula. The skin is pale and white. He cries constantly, sucks his fingers or searches frantically for something to suck. He is rigid and stiff when held; difficult to cuddle.

Laboratory studies, including blood counts, chemistries, stool and blood cultures, spinal fluid studies, x-rays and allergy studies, have been discouragingly normal. Following poor intake, the stools become semi-loose or loose, with secondary dehydration. Without supportive therapy, metabolic acidosis, with significant changes in blood chemistries, may occur.

Handling these infants has proven difficult. Sufficient nourishment, by supplementary parenteral feedings, gavage feedings, etc., must be supplied. The rapid collapse and shock-like state has responded to steroid therapy.

The most important phase of treatment is TLC; to make the infant feel loved, wanted and secure; to elicit some emotional response from the apathetic infant, and to calm the anxious infant. Bottles are never propped for feeding. Patient, careful feeding is stressed. The infant is held, rocked, talked to, cuddled and reassured. In short, the most important treatment is mothering.

As soon as the physical condition permits, placement in prospective adoptive homes or good foster homes is recommended.

In the case presentations which follow, the diagnosis of emotional disturbance was made by elimination. Careful and frequent clinical

examinations of the infant; laboratory studies, including complete blood counts and urinanalyses, spinal fluid cell counts, chemistries and cultures; blood chemistries; blood and stool cultures; x-ray studies; allergy tests, as well as tests for pancreatic dysfunction and phenylketonuria were performed.

CASE 1. Apathetic. This white male was born after a full term pregnancy, the mother's first. The mother, rated as low normal mentality, was not employed, but kept house for the family. She was unmarried. The serology was non-reactive, blood type A, rh factor, positive. She seemed withdrawn, seldom if ever smiled, and rarely talked with anyone. The pregnancy was uncomplicated, and she delivered spontaneously after a labor lasting 6 hrs. 11 mins. The history of the putative father is non-contributory.

The infant weighed 7 lb. 3 oz. at birth. No distress was reported. He was admitted to St. Vincent's at 4 days of age. Admission physical revealed a normal infant, weighing 6 lb. 11 oz.; measuring 50 cm. in length. He was fed Similac, the house formula and did fairly well for about three weeks. He was noted to be somewhat hypotonic, seldom cried, and had to be urged to eat during this period. He suddenly lost interest in eating, lost weight rapidly and failed rapidly. The stools became greenish and frequent following the weight loss. Blood studies, urinanalyses, stool cultures and x-rays of the chest and G.I. tract were normal. Various formulas were tried, but there was no difference noted. Gavage feedings and supplementary parental fluids were instituted when he stopped eating. Despite adequate supportive therapy, he continued to fail, until he presented a typical picture of marasmus. Fluids, including whole blood, and supportive therapy were continued. His course vacillated for 4 weeks, then gradually improved. At two months of age, he showed an interest in eating, cried when hungry and responded to mothering.

He progressed rapidly and was transferred to the regular nursery at 3 months of age. He continued to do well, but developed an upper respiratory infection with possible allergic bronchitis at 4 months of age. He recovered quickly and easily from the infection.

Psychological evaluation was obtained at 4½ months of age. The examiner felt he was a normal infant for his age. He was released to prospective adoptive at 5½ months of age.

CASE 2. Apathetic. This white male was admitted to St. Vin-

cent's at 2 weeks of age, remained there for nearly 3 months, when he was placed with prospective adoptive parents.

He was delivered after a 39 week pregnancy, the mother's first. There were no apparent complications, but the mother received no pre-natal care until the 7th month. The mother, a 25 year old secretary of normal intelligence, was very disturbed about this illegitimate pregnancy.

Following labor of 17 hrs. 30 mins., delivery was assisted with outlet forceps. No immediate complications were recorded. Birth weight was 5 lb. 9 oz. He was placed on Similac formula and was reported to be a feeding problem from the onset of feeding.

The putative father was described as 32 years old, with normal intelligence. He had diabetes. The information concerning the father's diabetes was not known to us until after the infant had been discharged.

Weight on admission was 5 lb. 10 oz.; length was 45 cm., with head and chest measurements in the 50th percentile. He appeared listless and apathetic, but the physical examination was within normal limits.

During the first few days he was given the house formula, Similac, which he took poorly, even with urging. He cried very little, even when stimulated. He was listless, his eyes were dull, he showed no emotion, appeared withdrawn and disinterested; and spent all of the time curled in a fetal position. Ten days after admission, he showed minimal dehydration; weight was 5 lb. 11 oz. Supplementary fluid therapy restored his hydration and a period of approximately 10 days of special TLC produced an apparent improvement in physical condition and mental outlook.

He was transferred to another nursery, at which time he stopped eating and failed rapidly. The stools became loose, but repeated cultures failed to produce pathogenic organisms. He responded to hydration, supportive therapy and TLC. After three weeks he showed more emotion than ever before, was eating vigorously and crying to be fed. When it was thought that he would do well, he was transferred to another nursery, but the same pattern of not eating, weight loss, loose stools, and rapid failure developed within three days.

He was returned for special care, hydrated and given supportive therapy and remained in the infirmary until his discharge on July 29, 1959.

He gained steadily and well for the several weeks prior to his discharge. There were no loose stools. He was released to prospective adoptive parents at the age of 3 months. At that time, he had a vigorous cry, ate well, smiled and cooed. His weight, which had been 6 lbs. on his last admission to the infirmary, was 7 lbs. 9 oz.

An inguinal hernia had been noted on his last admission to the infirmary. This was not visible again until the day of discharge. Because of his poor physical condition, his rapid failure when transferred from the infirmary, and the fact that the hernia was seen only once, repair was not considered. When it was seen on the discharge physical examination, it was suggested to the prospective adoptive parents that the infant remain at St. Vincent's until surgical repair of the hernia was done. They did not want to do this, and said they would have their own physician see the infant and arrange for the repair.

Laboratory studies at St. Vincent's were varied, including blood counts, with hemoglobin ranging between 11-16 gms; WBC's ranging from 13,450 to 7,850 with normal differentials. Stool cultures at St. Vincent's and the Philadelphia Department of Health laboratory were negative. Urinalyses, x-rays of the G.I. tract, stool trypsin and salt concentration tests in the sweat were all normal.

On 8-8-59, ten days after his discharge, he was admitted to another hospital with the complaint of diarrhea and vomiting. The parents stated that he had had several loose stools on the way home from St. Vincent's hospital. These had subsided the following day. Their physician had seen the infant the day after discharge, and could find nothing wrong. The hospital admission physical revealed mild dehydration, but was otherwise normal. Treatment was directed toward control of diarrhea, fluid and electrolyte balance. 80 cc of whole blood were given on the 3rd hospital day. On the 4th hospital day, he became cyanotic, comatose and expired several hours later. The autopsy revealed only a moderately dehydrated infant.

CASE 3. Apathetic. This white female was delivered on 4-23-59 and admitted to St. Vincent's on 4-28-59.

The mother was an unmarried 37 year old gravida; para O; a business college graduate, of normal intelligence. She had received adequate pre-natal care from a private physician. The history of the putative father was non-contributory.

The mother was first seen by the social case worker two days after she had delivered. The case worker reported that the mother was obviously upset and disturbed about the pregnancy, and could not discuss plans for the infant coherently at that time. The infant was placed at St. Vincent's until the mother could discuss and settle her problems. Maternal serology was non-reactive; blood type O; rh positive.

The duration of the pregnancy was 40 weeks. The time of labor is not known. Delivery was via low forceps, with no apparent complications. Birth weight was 7 lbs.; length 19½ ins. Physical examination on admission was not remarkable. Weight was 6 lb. 13 oz.

She was reported to be a quiet baby who seldom cried. She was a slow feeder from the time of admission. With much coaxing she would take her bottle. Vomiting was reported frequently, but there was no pattern to the vomiting nor did it get worse. During the first month, her weight ranged between 6lb. 10 oz. and 7 lb. 3 oz., which she weighed at the age of 1 month. She was a sad looking baby.

Laboratory studies were normal. Blood counts were normal. Eosinophile counts and smears were normal. Stool cultures for pathogenic organisms were negative.

Various formulas, including allergenic formulas, were tried, but the poor intake, lack of interest and vomiting persisted regardless of the type of formulas given.

TLC was stressed. She responded slowly to the mothering, but did eat better, ceased vomiting and became more interested. As soon as the mother had decided to release the infant for adoption, she was discharged to prospective adoptive parents on 6-20-59, at which time it was felt that she would do better in a family situation. Discharge weight was 8 lb. 7 oz.; length 59 cm.

CASE 4. Hyperactive. This white male infant was admitted after uncomplicated outlet forceps delivery, following a full term uncomplicated pregnancy. The mother was a 46-year-old grav. iii, para ii widow. She was reported to have low normal mentality, had left school after the 8th grade and was employed as a trimmer in a clothing factory. Her previous pregnancies had occurred 23 and 25 years before this. She was very disturbed and ashamed of this pregancy.

Serology was non-reactive, blood type A, rh positive. She had taken insulin, 40u daily for 9 years prior to this pregnancy. Careful study revealed no evidence of diabetes, and the insulin was discontinued early in pregnancy, with no difficulties after it was stopped. There was no history on the putative father.

Delivery was spontaneous after labor lasting 2 hours. There were no complications. Birth weight was 9 lb. 1 oz.

Physical examination on admission to St. Vincent's was within normal limits. Wt. 8 lb. 14 oz.; length 50 cm. He was given Similac, but reported to cry and fuss frequently. Frequent stools, occasionally with fluid were reported. Various formulas were tried, as were antispasmodics, but the pattern remained the same. An ENT consultation was obtained, since some of the nurses reported he had difficulty swallowing. The consultant could find nothing wrong. Mild sedation, Elixir Phenobarbital with much TLC was given. He did improve, slept better and was released to prospective adoptive parents at 3 months of age; weight 12 lb. 6 oz., length 61 cm.

CASE 5. *Hyperactive.* This white female was admitted to St. Vincent's at the age of 4 days, after a full term, uncomplicated pregnancy. The mother was an unmarried, 18-year-old grav. i, para O, high school graduate of average mentality.

The mother was disturbed emotionally, not by the pregnancy, but because of a very unhappy home situation. She was reported to be a possible pre-eclamptic and received a diuretic, Diamox, periodically during the pregnancy.

Labor lasted 8 hours, delivery was spontaneous, with no complications. The admission physical was not remarkable. Weight was 6 lb., length was 49 cm.

The baby screamed most of the time and moved constantly in the bassinet. She slept very little. The stools contained mucous, but there was no diarrhea. Laboratory studies were within normal limits, including studies for allergy. Stool cultures were negative. X-rays of the G.I. tract were negative. Because of the pattern of cat naps, screaming, and crying, spitting up of formulas, which persisted, an EEG was obtained. This was normal. Sedatives, antispasmodics, and tranquillizers were tried, but did not help. It required one nurse to care for this baby or she would scream until the entire nursery was upset. There was some improvement dur-

ing the last month of her stay, inasmuch as she slept better at night.

The mother would not release the infant for adoption, and could not take the infant until she was 5½ months of age. At that time, the mother married and took the baby. The infant smiled, grasped, rolled over and sat well when propped. She was alert, but still fussy.

CASE 6. Hyperactive. This white male was delivered after full term uncomplicated pregnancy, the mother's first. The mother was a 22-year-old office worker of normal mentality and unmarried. She was not disturbed during the pregnancy. Serology was non-reactive. She delivered spontaneously after labor of 18 hrs. 33 min. There were no complications. Birth weight was 7 lbs. There was no history of the putative father.

On admission to St. Vincent's at the age of 4 days, the infant was slightly jaundiced, weighed 7 lbs. and was 50 cm. long. He was given Similac and gained well. At the age of 3 weeks, he showed signs of hyperirritability and became difficult to feed. He vomited his feedings. His knees were raw. A papular rash developed over both cheeks. He would eat ravenously, but was never satisfied with his feedings. Formula changes, including allergenic formulas, made no difference. Addition of solid food did not satisfy him.

Blood counts were normal with hemoglobin between 9-11 gms; and WBC's between 9,050 and 15,950 with normal differentials. Stool cultures were negative. He calmed somewhat when given Elixir Donnatal before each feeding.

At the age of 9 weeks he was placed with prospective adoptive parents. He returned to St. Vincent's three weeks later, because the adoptive mother could not handle him. Both the infant and mother were getting increasingly upset.

He was in good physical condition and was placed in a second prospective adoption home a week after his re-admission. Apparently, he has adjusted well in this home.

DISCUSSION

The intimate relationship of an infant and mother has been stressed by many authorities. Physiologically and anatomically, the relationship of a mother and the child growing within her uterus is very close. Emotionally also, the mother and her child are very close. This close relationship begins at conception, not at the birth of the infant.

Pregnancy is a situation charged with implications and many pressures are exercised upon the pregnant woman from our culture. These pressures vary, depending on whether or not, the culture approves of the various factors of the pregnancy.

Increased activity has been observed in fetuses near term, when the mother was experiencing emotional stress. If the unborn fetus can reflect emotional stresses from the mother, the neo-natal infant should be able to reflect emotional stresses from the mother.

Infants express anxiety, stresses and tensions through various means; difficulties of feeding, spitting elimination activity, chiefly bowel functions; activity, over or under-activity; comfort and discomfort. These patterns have been noted in infants who have been deprived of mothering during the first few months of life. However, such patterns, when seen in newborn infants cannot be due to lack of mothering. The factors producing these patterns must arise before birth.

In an attempt to correlate the infants who presented problems with emotionally disturbed mothers, 100 records were pulled at random and carefully reviewed.

Questionaires regarding the mother; her past history; pre-natal care; intelligence; education; type of employment; family relationships; emotional status; and medications received, were used. The mothers were grouped as follows:

Emotionally disturbed: those who were noticeably upset during the pregnancy.

Not disturbed: those who demonstrated no evidence of emotional upset during the pregnancy.

Emotional status unknown or uncertain: those whose emotional status could not be determined with a reasonable degree of accuracy.

The infants were classified according to the type of problem they presented:

Physical: those infants who demonstrated sufficient evidence on physical examination or laboratory evidence to account for the problem, and whose difficulties ceased when the physical problem was adequately treated.

Physical plus: those infants for whom we could not demonstrate sufficient physical or laboratory evidence to account for the diffi-

culties, and whose difficulties did not disappear after the physical problem was adequately treated.

Psychological: those infants for whose problems no physical or laboratory evidence could be found.

The following statistics were obtained:

TABLE I

Mothers: 100		Infants: 100
Male—48		Female—52
44	White	46
2	Negro	42
2	White-Negro	2

TABLE II

Mothers		Infants					
		Male		Female		Total	
		No.	%	No.	%	No.	%
Emotionally Disturbed.							
38		24	63	14	37	38	100
	Problems	14	37	6	15	20	52
	Physical	2	5	2	5	4	10
	Physical Plus	9	23	4	10	13	34
	Psycho.	3	7.5			3	7.5
	No problems	10	26	8	21	18	48
Not Disturbed.							
38		13	34	25	66	38	100
	Problems	6	15	8	21	14	36
	Physical	3	8	6	15	9	23
	Physical Plus	3	8	2	5	5	13
	No problems	7	18	17	44	24	64
Unknown Emotional Status							
24		11	45	13	54	24	100
	Problems	1	4	3	13	4	17
	Physical			1	4	1	4
	Physical Plus	1	4	2	8	3	12
	No problems	10	41.5	10	41.5	20	83

Of the 100 records reviewed (Tables I & II):

38 mothers were emotionally disturbed. Of the 38 infants from

these mothers: 20 or 52% presented problems. 4 or 10.5% presented physical problems. 13 or 34% presented physical plus problems. 3 or 7.5% presented phychological problems. 18 or 48% presented no problems.

Disregarding physical problems, 41.5% of these infants presented problems for which we could not account on physical or laboratory basis or which persisted after the physical problem had ceased.

TABLE III

Types of Problems:		
Physical:		
	Male:	1—Mongolian (w) 1—post maturity and fetal pneumonia (w) 1—staph pneumonia (w) 1—bronchiolitis (w) 1—anemia (n)
	Female:	4—allergy to cow's milk (w) 1—pyelitis (w) 2—birth trauma: fractured clavicle, intracranial bleeding (w) 1—congenital heart disease. (n) 1—hydrocephalus (n)
Physical plus:		
	Male:	5—possible milk allergy and extreme fussiness (w) 1—possible milk allergy and extreme fussiness (w-n) 3—G. I. spasm, pylorospasm. (w) 1—birth trauma, mild facial paralysis and extreme fussiness. (w) 2—generalized rigidity, tremors and fussiness (w) 1—vomiting, hyperactivity and fussiness (n)
	Female:	5—possible mild allergy and extreme fussiness (w) 1—tremors with feeding problem (w) 2—tachycardia, cough, extreme fussiness (w) (w-n)
Psychological:		
	Male:	3—severe feeding problems, marasmus (w)

38 mothers were not disturbed. Of the 38 infants from these mothers: 14 or 36% presented problems. 9 or 23% presented physical problems. 5 or 13% presented physical plus problems. 24 or 64% presented no problems.

Disregarding physical problems 13% of these infants presented problems for which we could not account on physical or laboratory basis or which persisted after the physical problem had ceased.

24 mothers were of unknown emotional status. Of the 24 infants from these mothers: 4 or 17% presented problems. 1 or 4% presented physical problems. 3 or 12% presented physical plus problems. 20 or 83% presented no problems.

Disregarding physical problems 12% of these infants presented problems for which we could not account on physical or laboratory basis or which persisted after the physical problem has ceased.

SUMMARY

Our inability to discover sufficient physical or laboratory evidence to explain the problems presented by some of the infants at St. Vincent's Hospital has convinced us that there may be an emotional basis for the infant's difficulties.

41.5% of the infants delivered of emotionally disturbed mothers showed some emotional disturbance.

13% of the infants delivered of mothers who were not emotionally disturbed showed some emotional disturbance.

12% of the infants delivered of mothers whose emotional status was unknown or uncertain showed some emotional disturbance.

With adequate treatment of the emotionally disturbed pregnant woman and with the information that the mother was disturbed during the pregnancy, the physician caring for the infant born of such a mother may be able to treat the infant more intelligently.

CONCLUSION

Descriptions of two types of apparent emotional disturbances in newborn infants and in neo-natal infants have been presented, with an attempt to correlate such infants with mothers who were under emotional stress and tension during the pregnancy.

BIBLIOGRAPHY

1. Bakwin, Harry, M.D.; and Bakwin, Ruth, M.D.: Clinical Management of Behavior Disorders in Children, Philadelphia, Saunders, 1954.
2. Bakwin, Harry: Early Infantile Autism, Journal of Pediatrics, Vol. 45, Oct. 1954.
3. Child Psychiatry and the General Practitioner, Medical Times, July 1956.
4. Denhoff, Eric: The Syndrome of Cerebral Dysfunction, Jr. of Oklahoma State Medical Association, June 1959.
5. Gesell, A. L., and Ilg, F.: Feeding Behavior of Infants, Philadelphia, J. B. Lippincott Co., 1937.
6. McFarland, M. B., Ph. D., and Rhinehart, John B., M.D., The Development of Motherliness, Children, March-April, 1959.
7. Ribble, Margaret A., M.D.: The Rights of Infants, 1943.
8. Sutton, H. Eldon: Abnormal Amino Acid Metabolisms in a Case Suggesting Autism, Journal of Diseases of Children, July 1958.

70 Rittenhouse Place, Ardmore, Pa.

Post-Natal Head Injury as a Cause of Mental Defect

J. M. Berg, M.B., B.Ch., M.Sc.*
England

A vast literature has grown up around the subject of post-natal head injuries. Though some authors have referred in passing to the role of such injuries in the production of mental defect, little attention has been devoted specifically to this question. This is all the more surprising as children are particularly prone to trauma of all kinds; and parents, confused perhaps by the unexpected and frequently inexplicable catastrophe of having a mentally defective child, may well attach an undue significance to some such traumatic event. Such an explanation is readily acceptable as it is likely to relieve guilt feelings still commonly associated with any suspicion of a hereditary "taint". A consideration of the problem is therefore presented.

In the 10 years from 1949 to 1958 inclusive, 3 children have been admitted to the Fountain Hospital in whom post-natal cerebral trauma may have been of aetiological significance in relation to their mental defect. The Fountain Hospital admits children from London and Surrey, nearly all of whom are under 6 years on admission and of idiot or imbecile level. The average annual intake of patients is 90, so that the 3 children are among some 900 who have passed through the hospital in the period under consideration.

Findings in the 3 children are summarised in Table I.

Mental deterioration and epilepsy are occasional, though rare, sequelae of vaccination (Tredgold, 1952; Wilson, 1954). Such a course of events may have occurred in Case 1, though absence of clinical details of the post-vaccination state makes this uncertain. Nevertheless, the child was definitely epileptic and apparently retarded prior to his head injury. However, a defective child subjected to cerebral trauma may deteriorate still further after a head injury, and this seems a possibility here. Bowman and Blau (1949) and Peterson (1956) have emphasized that head injuries more commonly complicate already existing mental defect than actually produce it.

* Clinical Research Fellow, Fountain Hospital, London.

TABLE I

Findings in 3 mental defectives with head injuries

			1	2	3
SEX			Male	Male	Female
DATE OF BIRTH			30.9.1947	16.1.1948	7.8.1950
BIRTH WEIGHT			7 lbs. 13 ozs. (3.544 kg.)	8 lbs. 4 ozs. (3.742 kg.)	6 lbs. 5 ozs. (2.863 kg.)
FAMILY HISTORY OF MENTAL OR NEUROLOGICAL DISEASE			Nil	Nil	Mother's blood W.R. 6 Kahn +ive (repeated) after labour. 1st anti-syphilitic therapy post-natally
PREGNANCY AND LABOUR			Normal	Normal	Illegitimate. Normal
PRETRAUMATIC DEVELOPMENT			Considered normal till petit mal began at 6 months following vaccination. "Slightly retarded" at 1 year, without other abnormalities	Considered normal till 2½ months	Repeated investigations for syphilis -ive in 1st 5 months. Apparently normal till accident. Medical certificate of "perfect health" at 10 months
TRAUMATIC EVENT			Fell from mother's arms at 11 months, bumping head	Assaulted by father at 2½ months	Fell from high chair at 11 months
POST-TRAUMATIC SEQUELAE	Initial	Skull Fracture	Right parietal bone	Right parietal bone	Nil
		Coma	Absent	?	Present
		Other Findings	Pyrexia with fluctuant swelling over right parietal bone after 1 days. Responded to penicillin. Nil else found	Oedema of face and scalp. Bilateral subdural haematomata	Fixed, dilated pupils. Trismus. Rigid, extended limbs. Extensor plantars. No subdural haematoma on needling
	Later	Epilepsy	Continued petit mal	Grand and petit mal	Petit mal
		Other Findings	Nil	Mentally retarded. Head circumference at 2y months was 39.4 cm. (Normal = 45.7 cm., S.D. ± 1.40)	Gradual return to consciousness over several weeks. Spastic diplegia. Little progress in development
PRESENT OR ASCERTAINED TO PERTAIN	Chronological Age (in yrs)		5½	5½	1 11/12
	Developmental Age (in months)*		46	3 - 4	6
	Mental Status		Imbecile	Idiot	Imbecile
	Head Circumference (in cm.)**		51.4 (51.8, S.D. ± 1.47)	42.2 (50.4, S.D. ± 1.35)	47.2 (48.0, S.D. ± 1.32)
	Physical Signs		Nil	Blind. Spastic diplegia	Ptosis. Hemiparesis. Extensor plantars. Ankle clonus. Petit mal. No clinical or serological evidence of syphilis
ELECTRO-ENCEPHALOGRAM			At 11 years: Slightly abnormal in that normal frequencies are almost absent, and record consists of low voltage 3-a-second activity with occasional fast activity at about 20-a-second		At 9 years: Very abnormal. Record suggestive of diffuse brain injury, with suppression in right parieto-occipital area, possibly as result of large atraumatic lesion
SUBSEQUENT DEVELOPMENT			No significant change in next 6 years. Periodically restless and destructive	No significant change till death from dysentery at 6 years. Tuberous sclerosis unexpectedly found at autopsy	No significant change in next 7 years. Usually quiet and friendly

* On Vineland Social Maturity Scale. ** Normal values, in brackets, are those of Scammon and Harker (1956).

While the injury sustained by Case 1 was sufficient to produce a fractured parietal bone, the physical sequelae were slight and there was no period of unconsciousness. However, brain injury associated with mental defect is not necessarily accompanied by abnormal neurological signs. Further, Strauss and Savitsky (1934) have observed that significant intra-cranial injury can occur without loss of consciousness.

Clearly, it is impossible to ascribe the defect in our case solely to the trauma. But its possible contribution cannot be excluded because the child was neurologically abnormal before, or because of the absence of post-traumatic neurological signs or unconsciousness.

It would be hard to find a better history of cerebral trauma leading to gross mental and physical defects than in Case 2. However,

the tuberous sclerosis unexpectedly found at post-mortem (the brain did not become available for detailed study) may well have been the main cause of the defect. It is difficult to believe that the trauma played no part, particularly in view of the bilateral subdural haematomata, which Voris (1950) considers are usually associated with severe brain injury and may lead to spasticity, convulsions and lack of normal development.

In Case 3 also, the head injury is very suggestive as the cause of the subsequent mental and physical state. Here no skull fracture was found, but gross brain damage may occur without fracture of the skull (Fabian, 1956; Strich, 1956). The child may possibly have developed meningo-vascular syphilis from the mother, who had her first anti-syphilitic treatment post-natally, but the repeated absence of clinical and serological evidence of syphilis makes this highly improbable.

Fabian and Bender (1947), Bakwin (1949) and others have emphasised the importance of considering pretraumatic conditions in assessing the consequences of cerebral trauma. Apart from the possible significance of the known factors of vaccination in Case 1, tuberous sclerosis in case 2, and syphilis in Case 3, it is also possible that each of these children may, to some extent, owe their defect to other, unknown, factors. Case 2, for instance, was not suspected of having tuberous sclerosis in life, and, had he not come to autopsy, the presence of this condition may not have been realised. The role of the head injury in these children is therefore not conclusively established, but it seems a factor of increasing significance in each of the 3 cases successively.

It will be noted that in all 3 cases epilepsy followed the head injury, though in Case 1 petit mal was already present previously. It has been estimated that about 3% develop epilepsy after closed, and about 50% after penetrating, head injuries (Phillips, 1954). As intellectual deterioration may follow post-traumatic epilepsy (Blau, 1936; Bakwin, 1949; Fabian, 1956), and as such epilepsy may be delayed for months, and even years, after the trauma (Russell, 1942; Richardson, 1951), mental retardation consequent on the epilepsy may similarly be delayed. The prognosis on the mental state following on head injury should, therefore, be guarded for a considerable period.

DISCUSSION

Even assuming that all 3 of the present cases owed their defect

partly or wholly to head injury, its incidence as a causative agent of severe mental defect in childhood would only be 0.3% (3 out of 900 cases). This indicates that idiocy or imbecility is a rare consequence of head injury in childhood. Among others, Ireland (1877)*, Blau (1936), Russell (1942), Bakwin (1949), Penrose (1954) and Peterson (1956) have noted the rarity of severe mental defect consequent on post-natal cerebral trauma in the young. Though rare in later life also, it may be less unusual (Russell, 1942; Symonds, 1949; Richardson, 1951; Peterson, 1956). One probable reason for this is the generally accepted fact that children tolerate head injuries better than adults, and hence less often show serious consequences (Glaser and Shafer, 1932; Russell, 1933-4; Blau, 1936).

It seems likely that the main reason why post-natal cerebral trauma does not lead to severe mental defect more often is that brain injury extensive enough to produce such serious consequences is frequently fatal.

Less severe forms of post-traumatic intellectual impairment are commoner but more difficult to assess. Behaviour disturbances and personality changes following head injuries may reflect themselves as inattention, lack of concentration and indifference, with consequent poor results at school, at work, or on psychometric testing. A subject may therefore seem mentally retarded without having suffered what Blau (1936) calls a "primary intellectual defect". Ruesch (1944) considered that "about one half of all subjects suffering from head injury show slight intellectual defects". Newell (1937) studied the effect of head injury on 20 patients from 6 to 17 years old whom he saw at child guidance clinics. In 5 (25%), mental efficiency was impaired, as evidenced by poor school work, though as he points out, evidence for an actual change in mental ability was not convincing. Three of these 5 children had Stanford-Binet tests both before and after their injuries, with substantially similar results. Rowbotham et al. (1954) followed up children with head injuries by means of a questionnaire to the parents. The authors judged 8 of 82 to have an intellectual injury of sufficient degree as to be "likely to influence adversely the scholastic career of these children for the rest of their lives".

It is evident that the incidence of intellectual impairment following head injury varies with the criteria accepted for what consti-

* In passing, it is of interest to note that Ireland takes up the question of head injury not only as a cause, but also as a cure, of idiocy. He considers the latter "a very rare event, *even in upper class schools*" (italics mine). He quotes, disbelievingly be it said, the story of a certain Father Mabillon who "was an idiot till the age of twenty-six, when he had the good luck to fracture his skull, was trepanned and changed into a learned writer upon ecclesiastical history".

tutes such impairment and probably with the age range of the sample under consideration. Studies of institutionalised populations of mental defectives, including both higher grades of defect and older patients than in this series, indicates that even then post-natal head injury is not a common cause of mental defect. In the Colchester survey, (Penrose, 1938) reported 12 of 1,280 cases (0.9%) in whom such trauma was aetiologically important. Investigating 1,000 consecutive admissions to a large New York State institution for mental defectives, Boldt (1948) concluded that "approximately 1.5% of institutionalised mental defectives can be considered to be the result of post-natal cerebral trauma."

SUMMARY

Among some 900 low-grade mentally defective children admitted to the Fountain Hospital from 1949 to 1958 inclusive, were 3 (0.3%) in whom post-natal cerebral trauma may have been aetiologically significant. Findings in the 3 chidren are tabulated and discussed.

It is concluded that idiocy or imbecility is a rare consequence of head injuries in childhood. Published surveys of institutionalised populations of mental defectives, including both higher grades of defect and older patients than in the present series, indicate that even then post-natal head injury is not a common causative agent.

ACKNOWLEDGEMENTS: I am indebted to Dr. J. Foley for the E.E.G. reports on Cases 1 and 3, to my colleagues at the Fountain Hospital for helpful suggestions, and to the staff of the Psychology Department, Fountain Hospital, for the psychological assessments of the 3 children.

REFERENCES

Bakwin, H., J. Pediat., (1949), 34, 371.
Blau, A., Arch. Neurol. Psychiat. (Chicago), (1936), 35, 723.
Boldt, W. H., Amer. J. ment. Defic., (1948), 53, 247.
Bowman, K. M. and Blau, A., *in* Injuries of the brain and spinal cord and their coverings. (Edited by S. Brock). (1949) 3rd ed., Chapter 13, Baillière, Tindall & Cox, London.
Fabian, A. A., J. nerv. ment. Dis., (1956), 123, 428.
Idem and Bender, L., Amer. J. Orthopsychiat., (1947), 17, 68.
Glaser, M. A. and Shafer, F. P., J. Amer. med. Ass., (1932), 98, 271.
Ireland, W. W., Idiocy and imbecility, (1877), Churchill, London.
Newell, H. W., Med. Clin. N. Amer., (1937), 21, 1335.
Penrose, L. S., A clinical and genetic study of 1,280 cases of mental defect. (1938), p. 41. H.M.S.O., London.
Idem, The biology of mental defect. (1954), Revised ed., p. 197. Sidgwick & Jackson, London.
Peterson, B. H., Med. J. Aust., (1956) i, 689.
Phillips, G., J. Neurol. Neurosurg. Psychiat., (1954), 17, 1.
Richardson, J. C., Canad. med. Ass. J., (1951), 64, 414.
Rowbotham, G. F., Maciver, I. N., Dickson, J., and Bousfield, M. E., Brit. med. J., (1954), i, 726.
Ruesch, J., Amer. J. Psychiat., (1944), 100, 480.
Russell, W. Ritchie, Trans. med.-chir. Soc. Edinb., (1933-4), 113, 129.
Idem, Brit. med. J., (1942) ii, 521.
Strauss, I., and Savitsky, N., Arch Neurol. Psychiat. (Chicago), (1934), 31, 893.
Strich, S. J., J. Neurol. Neurosurg. Psychiat., (1956), 19, 163.
Symonds, C. P., *in* Injuries of the brain and spinal cord and their coverings. (Edited by S. Brock). (1949), 3rd ed., Chapter 4, Baillière, Tindall & Cox, London.
Tredgold, A. F., A textbook of mental deficiency (amentia). (1952) 8th ed., p. 268. Baillière, Tindall & Cox, London.
Voris, H. C., Arch. Surg. (Chicago), (1950), 60, 906.
Westropp, C. K., and Barber, C. R., J. Neurol. Neurosurg. Psychiat., (1956), 19, 52.
Wilson, S. A. Kinnier, Neurology. (Edited by A. N. Bruce), (1954), 2nd ed., p. 83. Butterworth, London.

Fountain Hospital, Tooting Grove, London, S. W. 17, England

Dietary Management of Phenylketonuria with Lofenalac ®

FRANK L. LYMAN, M.D.
JULIA K. LYMAN, R.N.
INDIANA

IN 1934 the syndrome known as phenylketonuria was discovered by a Norwegian physician and biochemist, Doctor Asbjorn Fölling.[1] The dietary management of this condition represents one of the first inroads of medicine in the prevention of mental deficiency.

Phenylketonuria is a hereditary error in the metabolism of the amino acid, phenylalanine. It is characterized by high blood phenylalanine levels and the presence of phenylpyruvic acids and other breakdown products of phenylalanine in the urine. This error is due to a deficiency of the hepatic enzyme, phenylalanine hydroxylase, which normally converts phenylalanine to tyrosine.[2, 3]

Phenylketonuria is a rare disease occurring about once in every 20,000[4] to 25,000 births.[5] Approximately one in every 70 persons in the United States is a carrier. All races are affected, but the incidence appears to be lower among the Negro, Jewish and Japanese populations.

DIAGNOSIS

Phenylketonuric children appear to be normal at birth and the disease cannot be detected at this time. At the age of one to six weeks the condition generally can be diagnosed through a simple ferric chloride urine or wet diaper test. Elevated blood phenylalanine levels (the normal is 1 to 3 mg. per cent) are confirmatory of phenylketonuria. Carriers may be detected by a phenylalanine tolerance test.

Untreated phenylketonuric children progressively develop a mental deficiency. These children rarely develop an I.Q. above 50. Many of these children have disagreeable and schizoid-like personalities. Approximately 80 per cent have electroencephalographic abnormalities, and eczematoid rashes and convulsions are common.[6] Characteristically, phenylketonuric children are more blond than their parents or normal siblings.

Fölling and many other investigators suggested that a low phenylalanine diet might be of value in the treatment of phenylketonuria. Armstrong[7] and Bickel,[8] and Woolf, working dependently, were the first investigators to prepare low phenylalanine diets. Armstrong's diet was essentially a mixture of amino acids, while Bickel's preparation consisted of a low phenylalanine protein hydrolysate from which most of the phenylalanine had been removed. Since then, numerous investigators[9-15] have confirmed their findings that low phenylalanine diets, if instituted early enough, will prevent mental retardation.

It is necessary to start the diet at an early age, preferably before three months, if normal mentality is to be maintained. Some benefit may be obtained in older children from the standpoint of improvement in personality, rashes and convulsions, although normal mentality probably cannot be regained. Centerwall[16] has reported on one patient with a D.Q. of 32 who was detected at the age of three years. After two years of treatment with a low phenylalanine diet, his D.Q. had risen to 60. Generally speaking, however, the improvement in intelligence in older children has not been so dramatic.

A few phenylketonuric children have been found with normal I.Q.'s and the question has arisen as to whether these children should be treated with a low phenylalanine diet. Bickel[17] reported on a six-year-old child that had a normal I.Q. at the age of six, but who was definitely retarded two years later. Bickel feels that this child should have been given a low phenylalanine diet.

PHENYLALANINE BLOOD LEVELS

Most investigators feel that phenylalanine blood levels of phenylketonurics should be kept within the normal range of 1-3 mg. per cent, although some investigators permit somewhat higher levels.

Since phenylalanine is an essential amino acid, diets cannot be completely deficient in this amino acid and still be nutritionally adequate. Generally, from 15 to 40 mg. of phenylalanine per kg. of body weight per day are permitted. Borofsky,[18] however, has reported two children that have been maintained with normal phenylalanine blood levels on diets containing 50 to 70 mg. of phenylalanine per kg. of body weight. Frequent blood phenylalanine determinations are important in determining the effectiveness of the low phenylalanine diet. Urine tests cannot be used to determine dietary effectiveness, since phenylpyruvic acid is found in the urine only when the phenaylalanine blood levels reach 12-15 mg. per cent.

LOW PHENYLALANINE DIETS

Phenylalanine is a ubiquitous amino acid and amounts to about 4 to 6 per cent of all protein, regardless of the source. Since it is impossible to devise a diet deficient in phenylalanine that is nutritionally adequate from normally occurring foods, the basis of all low phenylalanine diets is a commercially prepared low phenylalanine food. Two such products are commercially available in the United States. Ketonil®* is a powder made from casein hydrolysate from which most of the phenylalanine has been removed and amino acids, mineral salts, and choline chloride added. Ketonil requires the addition of sugar and vegetable oil to form the basis diet. The other product is Lofenalac®,** a complete food low in phenylalanine, which may be used as a powder mixed with foods and as a beverage when mixed with water.

THE LOW PHENYLALANINE DIET WITH LOFENALAC

Lofenalac is essentially a protein hydrolysate powder from which most of the phenylalanine has been removed. Methionine, tyrosine and tryptophan, vegetable fat, Dextri-Maltose,® minerals and vitamins are added by the manufacturer to make a complete food, low in phenylalanine. Certain other foods (mostly fruits and vegetables) low in phenylalanine may be permitted in addition to the basic amount of Lofenalac.

Most investigators agree that, initially, the phenylalanine intake should be severely restricted until blood levels are normal. For this reason, the initial diet should consist solely of Lofenalac beverage. Babies generally adjust well to this product. Older children with more varied tastes, coupled with their retardation, may require some patience before they adjust to the diet. The quantities fed should be comparable to the amount of cow's milk in the usual infant formulas.

One household measuring cup containing eight ounces (by volume) of the powder added to a quart of water makes approximately 32 ounces of beverage. Each ounce of the beverage contains 20 calories. The caloric and protein values are listed in Table I.

* Marketed by Merck Sharp & Dohme.
** Marketed by Mead Johnson & Company.

The severely restricted low phenylalanine diet should be continued only as long as it is necessary to lower the phenylalanine blood levels to normal. If continued for a longer period, the phenylalanine blood levels may rise again due to catabolism of the phenylketonuric's own body protein.

To make dietary calculations and exchanges simpler, the phenylalanine "Equivalent" was devised. The phenylalanine Equivalent was arbitrarily taken to equal 15 mg. of phenylalanine. This Equivalent is used throughout the diets presented in this article.

The amount of phenylalanine in the diet to maintain the phenylalnine blood levels at normal, varies from 15-70 mg. of phenylalanine per kilogram of body weight. Table II shows the amount of phenylalanine permitted in diets for children of various weights at the 25 mg. per kilogram level of intake. The amount of phenylalanine in Equivalents permitted beyond that contained in the Lofenalac, is also listed. These allow minimal amounts of protein in the diet. If possible to allow more than 25 mg.of phenylalanine per kilogram of body weight, better protein balance will be obtained.

A typical diet for a 20 pound baby is:

	Equivalents from Lofenalac	*Additional Equivalents*
Breakfast		
Lofenalac Beverage, 8 ounces	2	
Pablum® Rice Cereal, 4 level tablespoons		2
Lunch		
Lofenalac Beverage, 8 ounces	2	
Strained Applesauce, 10 tablespoons		1
Strained Green Beans, 3 tablespoons		2
Deca-Vi-Sol®, 0.6 cc.		0
Supper		
Lofenalac Beverage, 8 ounces	2	
Strained Pears, 10 tablespoons		1
Chopped Squash, 6 tablespoons		1
Bedtime		
Lofenalac Beverage, 8 ounces	2	
	8	7

In older children, this beverage may be concentrated to 40 calories per ounce (one 16 ounce measuring cup to a quart of water) or used as a powder mixed with other foods, for which certain recipes have been developed. It is important, however, that the basic amount prescribed be fed each day. It is equally important that the total number of phenylalanine Equivalents not exceed that calcu-

lated. The diet must be strictly adhered to if normal nutrition and mentality is to be obtained. Supplemental vitamins and iron are recommended.

Lofenalac in various forms has a distinctive taste which is difficult to mask. This flavor is intensified by overheating or browning. Phenylketonuric children soon become used to the taste and readily accept it. Tomatoes and pineapple will effectively mask the taste. Suggestions for various spices are also given.

It is difficult to formulate recipes that are low in phenylalanine, since milk, eggs and flour contain too much phenylalanine to be used. Lofenalac beverage (either single or double strength) may be substituted for milk in any recipe. Flour substitutes such as India Gum and Cellu® Celluose Flour may be used. Certain other substances contain little or no phenylalanine and may be used in any quantity. These are:

- Corn Oil
- Peanut Oil
- Olive Oil
- Granulated Sugar
- Powdered Sugar
- Brown Sugar
- Corn Starch
- Hard Candy
- Carbonated Beverages

The amount of phenylalanine in any food can be easily calculated if the protein content is known. Phenylalanine accounts for about 5 per cent of all protein. The phenylalanine values in Table III were calculated from Bowes and Church: *Food Values of Portions Commonly Used*, Eighth edition. The number of Equivalents in any portion can be obtained by taking 5 per cent of the amount of protein and dividing the result by 15.

TABLE I—CALORIES, PROTEIN AND PHENYLALANINE SUPPLIED BY TYPICAL QUANTITIES OF LOFENALAC

LOFENALAC	*Approx. Calories*	*Approx. Protein Gm.*	*Average Phenylalanine Mg.*	*Phenylalanine Equivalents*
100 Gm. (2/3 cup)	450	15	80	5
1 packed level measure (9.5 Gm.)	43	1.4	7.5	½
1 standard 8 ounce measuring cup (150 Gm.; 16 packed level measures)	680	22	120	8
1 fl. oz. of normal dilution*	20	0.6	3.8	¼
4 fl. oz. of normal dilution*	80	2.4	15	1
8 fl. oz. of normal dilution*	160	5	30	2
32 fl. oz. of normal dilution*	640	19	120	8

* 1 packed level measuring scoop to 2 fl. oz. water; or 1 level 8 oz. measuring cup to a quart of water.

TABLE II — TYPICAL QUANTITIES OF LOFENALAC AND OTHER FOODS IN TYPICAL DIETS

25 MG PHENYLALANINE PER KILOGRAM BODY WEIGHT

Body Weight	*Total Phenylalanine Allowed*	*Basic Lofenalac "Diet,"* Powder*	*Approx. Calories from Lofenalac*	*Phenylalanine from Lofenalac*	*Equivalents from Lofenalac*	*Other Foods Permitted Daily†*
10 lb.	110 mg.	⅞ cup	595	105 mg.	7	None
12 lb.	132 mg.	1 cup	680	120 mg.	8	1 Equivalent
14 lb.	154 mg.	1 cup	680	120 mg.	8	2 Equivalents
16 lb.	176 mg.	1 cup	680	120 mg.	8	4 Equivalents
18 lb.	198 mg.	1 cup	680	120 mg.	8	5 Equivalents
20 lb.	220 mg.	1 cup	680	120 mg.	8	7 Equivalents
24 lb.	244 mg.	1 ⅛ cups	765	135 mg.	9	8 Equivalents
28 lb.	308 mg.	1 ¼ cups	850	150 mg.	10	10 Equivalents
32 lb.	352 mg.	1 ½ cups	1020	180 mg.	12	11 Equivalents
40 lb.	440 mg.	1 ¾ cups	1190	210 mg.	14	15 Equivalents
48 lb.	528 mg.	2 cups	1360	240 mg.	16	17 Equivalents
60 lb.	660 mg.	2 cups	1360	240 mg.	16	21 Equivalents

* "Cup" refers to standard 8 ounce measuring cup (150 Gm. Lofenalac powder; equal to 16 packed level measuring scoops).

† Phenylalanine calculations are based on supplying approximately 11 mg. of phenylalanine per pound of body weight (25 mg. per kilogram) to provide for normal growth and development. One equivalent equals approximately 15 mg. of phenylalanine. One ounce of Lofenalac formula in normal dilution (20 calories per fl. oz.) contains 3.5 mg. of phenylalanine. Note: It is important that the level of Phenylalanine for the diet be carefully determined by frequent Blood phenylalanine levels.

TABLE III — PHENYLALANINE CONTENT OF VARIOUS FOODS

Food	*Approximate Measure*	*Phenylalanine Content*	*No. of Equivalents*
	Cereals		
Pablum® Mixed Cereal	1 level tablespoonful	18 mg.	1
Pablum Oatmeal Cereal	1 level tablespoonful	19 mg.	1
Pablum Barley Cereal	1 level tablespoonful	13 mg.	1
Pablum Rice Cereal	2 level tablespoonfuls	16 mg.	1
Rice Krispies®	½ cup	40 mg.	3
	Fruits		
Banana, raw	1 small	60 mg.	4
Cherries, maraschino	8 cherries	15 mg.	1
Apple	1 small	15 mg.	1
Cranberry jelly	1 rounded tablespoonful	trace	0
Applesauce, canned	½ cup, sweetened	15 mg.	1
Fruit cocktail	5 tablespoon, fruit and juice	15 mg.	1
Orange	1 small	45 mg.	3
Honeydew melon	¼ of a 5" melon	25 mg.	2
Peaches, canned	1 ½ halves	15 mg.	1
Olives, green	3 large	15 mg.	1
Pears, canned	2 halves	15 mg.	1
Pineapple, raw	½ cup	15 mg.	1
Pineapple, canned, crushed	½ cup	25 mg.	2
Raisins, dried	1 ½ tablespoonfuls	15 mg.	1
Rhubarb	½ cup	15 mg.	1
	Fruits (Infant)		
Applesauce, strained	10 tablespoonfuls	15 mg.	1
Applesauce, junior	10 tablespoonfuls	15 mg.	1
Apricot-Applesauce, strained	7 tablespoonfuls	15 mg.	1
Apricot-Applesauce, junior	7 tablespoonfuls	15 mg.	1
Peaches, strained	4 tablespoonfuls	15 mg.	1
Peaches, junior	4 tablespoonfuls	15 mg.	1
Pears, strained	10 tablespoonfuls	15 mg.	1
Pears with Pineapple, strained	7 tablespoonfuls	15 mg.	1
Pears with Pineapple, junior	7 tablespoonfuls	15 mg.	1
Plums with Tapioca, strained	5 tablespoonfuls	15 mg.	1
Plums with Tapioca, junior	7 tablespoonfuls	15 mg.	1
	Fruit Juices		
Tomato juice, canned	1 fluid ounce	15 mg.	1
Apple juice, canned	8 fluid ounces	15 mg.	1
Blueberry juice	10 fluid ounces	15 mg.	1
Grape juice	2 fluid ounces	15 mg.	1
Lemon juice	3 tablespoonfuls	15 mg.	1
Orange juice, fresh	1 ½ fluid ounces	15 mg.	1
Lime juice	3 tablespoonfuls	15 mg.	1
Orange juice, canned	1 ½ fluid ounces	15 mg.	1
Grapefruit juice	1 ½ fluid ounces	15 mg.	1
Pineapple juice	3 fluid ounces	15 mg.	1
Prune juice	3 fluid ounces	15 mg.	1

Food	*Approximate Measure*	*Phenylalanine Content*	*No. of Equivalents*
	Meats and Eggs (For comparison)		
Bacon	1 medium strip	90 mg.	6
Deviled meat	1 rounded tablespoonful	140 mg.	9
Hamburger	1 ounce	230 mg.	15
Meat gravy	1 tablespoonful	15 mg.	1
Frankfurter	1 average	350 mg.	23
Chestnuts	2 small	15 mg.	1
Egg	1 medium	300 mg.	20
Soy sauce	1 tablespoonful	trace	0
Worcestershire sauce	1 tablespoonful	15 mg.	1
	Soups		
Asparagus, creamed Campbell's	1/3 can	110 mg.	7
Beef Noodle, Campbell's	1/3 can	165 mg.	11
Chicken Gumbo, Campbell's	1/3 can	95 mg.	6
	Vegetables		
Beets	½ cup diced	40 mg.	3
Cabbage, raw	½ cup shredded	35 mg.	2
Carrots, cooked	½ cup diced	25 mg.	2
Celery, raw	1-8 in. outer stalk	25 mg.	2
Cucumber, raw	1/3 medium	15 mg.	1
Lettuce	2 small leaves	15 mg.	1
Onions, chopped, raw	3 tablespoonfuls	15 mg.	1
Parsnips, cooked	½ cup	40 mg.	3
Potato, baked	1 medium	120 mg.	8
Pickles	3 small (2" x 5/8")	15 mg.	1
Radish	3 small	15 mg.	1
Rice, cooked	1/6 cup	30 mg.	2
Squash, summer, cooked	1/2 cup	30 mg.	2
Tomato catsup	1 tablespoonful	15 mg.	1
Turnips, cooked	2/3 cup diced	40 mg.	3
	Vegetables (Infant)		
Green beans, junior	1 ½ tablespoonfuls	15 mg.	1
Green beans, strained	1 ½ tablespoonfuls	15 mg.	1
Beets, strained	2 tablespoonfuls	15 mg.	1
Carrots, strained	3 tablespoonfuls	15 mg.	1
Carrots, chopped	3 tablespoonfuls	15 mg.	1
Spinach, creamed strained	1 ½ tablespoonfuls	15 mg.	1
Spinach, creamed junior	1 ½ tablespoonfuls	15 mg.	1
Squash, strained	3 tablespoonfuls	15 mg.	1
Squash, chopped, junior	6 tablespoonfuls	15 mg.	1
Sweet potatoes, strained	2 tablespoonfuls	15 mg.	1
	Miscellaneous		
Beef tallow	2 tablespoonfuls	15 mg.	1
Butter	3 level tablespoonfuls	15 mg.	1
Chocolate syrup	½ ounce	17 mg.	1
Cocoa (dry)	1 ½ level tablespoonfuls	15 mg.	1
Crackers, Barnum's Animal	3 crackers	15 mg.	1
Crackers, cheese	1 cracker	15 mg.	1
Crackers, Saltines	1 cracker	15 mg.	1
Enriched wheat flour	1 level tablespoonful	40 mg.	3
French dressing	3 tablespoonfuls	15 mg.	1
Kitchen Bouquet®	1 tablespoonful	trace	0
Orange ice (water)	½ cup	20 mg.	1
Wafers, Nabisco® sugar	3 small wafers	15 mg.	1
White bread	1 slice	100 mg.	7
Whole milk	1 fluid ounce	50 mg.	3

The phenylalanine values in table three are calculated from Bowes and Church, *Food Values of Portions Commonly Used*. This book is an excellent source of protein values. The phenylalanine values are easily calculated from the protein values on the basis that all protein material contains approximately 5% phenylalanine.

SUMMARY

Phenylketonuria is a hereditary inborn error in the metabolism of phenylalanine that can be successfully managed with a special dietary preparation, low in phenylalanine.

ACKNOWLEDGMENT

Our sincere appreciation is expressed to Mrs. Anna DePlanter Bowes for use of the data from *Food Values of Portions Commonly Used*.

NOTE

Recipes and flavoring instructions referred to in this paper are available from the Authors as a supplement to the reprint.

REFERENCES

1. Folling, A.: Uber Ausscheidung von Phenylbrenztraubensaure in den Harn als Stoffwechselanomalie in Verbindung mit Imbezillitat., Ztschr. f. physiol. Chem. *227*:169 (1934).
2. Jervis, G. A.: Phenylpyruvic Oligophrenia Deficiency of Phenylalanine-Oxidizing System. Proc. Soc. Exper. Biol. & Med. *82*:514 (1953).
3. Udenfriend, S., and Cooper, J. R.: The Enzymatic Conversion of Phenylalanine to Tyrosine, J. Biol. Chem. *194*:503 (1952).
4. Lyman, F. L.: Phenylketonuria, New York J. Med. *58*:3653-3656 (Nov. 15) 1958.
5. Jervis, G. A.: Genetics of Phenylpyruvic Oligophrenia: (Contribution to Study of Influence of Heredity on Mental Defect), J. Ment. Sc. *85*:719 (July) 1939.
6. Jervis, G. A.: Phenylpyruvic Oligophrenia: Introductory Study of Fifty Cases of Mental Deficiency Associated with Excretion of Phenylpyruvic Acid, Arch. Neurol. & Psychiat. *38*:944 (1937).
7. Armstrong, M. D., and Tyler, F. H.: Studies on Phenylketonuria. I. Restricted Phenylalanine Intake in Phenylketonuria, J. Clin. Invest. *34*:565-580 (April) 1955.
8. Bickel, H., Gerrard, J., and Hickmans, E. M.: Influence of Phenylalanine Intake on Phenylketonuria, Lancet *2*:812-813 (Oct. 17) 1953.
9. Lotti, F.: Early Results of the Use of a Diet Free of Animal Protein in Therapy of Phenylketonuria, Clin. Pediat. *39*:917-924 (Dec.) 1957.
10. Gruttner, R.; Muller, F., and Wallis, H.: Evaluation of the Success of Dietetic Treatment with Casein Hydrolysate Low in Phenylalanine in Phenylpyruvic Oligophrenia, Monatsschr. Kinderh. *106*:41-45 (Feb.) 1958.
11. Hsia, D. Y.; Knox, W. E.; Quinn, K. V., and Paine, R. S.: A One-Year, Controlled Study of the Effect of Low-Phenylalanine Diet on Phenylketonuria, Pediatrics *21*:178-202 (Feb.) 1958.
12. Woolf, L. I.; Griffiths, R.; Moncrieff, A.; Coates, S., and Dillistone, F.: The Dietary Treatment of Phenylketonuria, Arch. Dis. Childhood *33*:31-45 (Feb.) 1958.
13. Meyer, H.; Mertz, E. T.; Stadler, H. E.; Leland, H., and Calandro, J.: Psychometabolic Changes in Phenylketonuria Treated with Low-Phenylalanine Diet, A.M.A. Arch. Int. Med. *101*:1094-1103 (June) 1958.
14. Karle, I. P.; O'Donnell, M. M., and Ehmke, D. A.: Enzyme Deficiency Diseases. IIa. A Phenylalanine-Restricted Diet, J. Am. Dietet. A. *34*:1051-1052 (Oct.) 1958.
15. Horner, F. A., and Streamer, C. W.: Phenylketonuria Treated from Earliest Infancy; Report of Three Cases, A.M.A. Am. J. Dis. Child. *97*:345-347 (March) 1959.
16. Centerwall, W. R.: Scientific Exhibit, presented at the A.M.A. Meeting, San Francisco, California, June 23-27, 1958.
17. Bickel, Horst, Universitats Kinder Klinik, Marburg-Lahn, West Germany: Personal communication.
18. Borofsky, Beatrice, St. Christopher's Hospital, Philadelphia, Pennsylvania: Personal communication.

2404 *Pennsylvania St.*

Evansville, Indiana.

Pediatric Conference . . .

THE ROOSEVELT HOSPITAL, NEW YORK

May 20, 1959

EDMUND N. JOYNER, III, M.D., *Chief of Pediatrics, Presiding*

DR. JOYNER: One of the most bothersome and perennial problems that every pediatrician has is to know when and whether to advise the removal of tonsils and adenoids. Actually at times the pressure from parents and particularly from grandparents to remove tonsils and adenoids, gets out of hand, especially where preschool and nursery school children are concerned. Yet, viewing the literature, there are very few truly controlled studies on the efficacy of this procedure and like any other operation, it is not without some danger.

There has been a tendency on the part of most pediatricians in recent years to be more conservative. It requires more of a consideration now than simply having tonsils.

In an attempt to bring to you the most up-to-date thinking on this subject, we are honored to have a panel of outstanding otolaryngologists and pediatricians present: Dr. Milton Levine, Associate Professor of Pediatrics, Dr. James Moore, Associate Professor of Clinical Surgery (Otolaryngology) and Chief, Division of Otolaryngology, both of New York Hospital-Cornell University Medical Center and Dr. Clark Grove, Chief of Otolaryngology Service, Roosevelt Hospital.

I'd like to start by asking Dr. Moore if he would shortly review the anatomy, histology and the functions of tonsils and adenoids.

DR. MOORE: Thank you, Dr. Joyner. I think I can summarize this very briefly as far as the anatomy is concerned. The tonsils are lymphoid structures, and we usually think of tonsils as the faucial tonsils or the ordinary tonsils; we have the so-called adenoids; and then, we have also the lingual tonsils. I think it is very important to understand that these structures have different histology. For instance, the faucial tonsils are composed of lymphoid substance with germinal centers and some 18 or 20 deep crypts. These crypts

are lined by stratified squamous epithelium and repeated infections or electro-coagulation or partial tonsillectomy are apt to block these crypts and lead to cysts, retention, multiple abscesses, and so forth, so that actually certain types of interference with the faucial tonsils may do more harm than good.

I'm sure you're familiar with the so-called Weber's glands, which are salivary-like glands located outside the tonsillar capsule, usually in the area of the superior pole. There is a great deal of discussion as to whether or not these salivary glands or mucous glands drain into the crypts. To the best of my knowledge, based on a considerable number of histological sections, I do not believe they do. You will hear arguments on both sides. The main importance of these mucous glands is that they are possibly closely related to the origin of peritonsillar abscess.

Now, let us turn to the so-called adenoids. From a practical standpoint, the most important problems regarding adenoids are: first, by their main "bulk" when they are infected and hypertrophied, and hyperplastic, they block the eustachian tubes. Here we have a source of eustachian tube obstruction and ascending middle-ear infection, and the various sequellae. I think we often forget the histology of the adenoids. The adenoid tissue has numerous crypts, but these crypts are lined by a pseudo-columnar ciliated epithelium and with numerous mucous glands.

In many instances, these mucous glands and the secretions from the mucous glands cause almost as much trouble as the mechanical blockage itself, because, under certain circumstances, with infection, with allergy, and under other conditions, this mucus becomes very thick and tenacious. This can accumulate in the mouth of the eustachian tubes and may cause blockage and subsequent infection. You do not necessarily have to have mechcanical blockage from enlarged adenoids to get middle-ear infections. The accumulation of mucus, whether it is from the adenoids or from an infected maxillary sinus, is just as important. The lingual tonsils, particularly after tonsils and adenoids are removed, may become hypertrophied and here we may have a chronic follicular type of infection.

Dr. Joyner, I think that covers, from the practical standpoint, the things I would like to mention.

DR. JOYNER: Now, Dr. Moore, what have we got tonsils and adenoids for? What is their function? I mean why do we start off with them? Why not take them all out?

DR. MOORE: The function of the tonsils and adenoids will pri-

marily have to do with resistance to infections. In this role, as a part of the reticulo-endothelial system, you have a barrier to infection and a means of obtaining immunity to infection located in a very critical area which is subjected to these inflammatory infections. I personally believe that in the early period this immunity and the resistance we get from repeated infections occurs primarily during the first three or four years. After that with severe infection, abscess formation, chronic follicular type infection, we may have enough infection to counter-balance any function that the tonsils or adenoids may have from the standpoint of immunity.

DR. JOYNER: In other words, you think until the tonsils are knocked out of commission by repeated infections they serve a very useful purpose?

DR. MOORE: Yes.

DR. JOYNER: Dr. Grove, do you agree essentially with that?

DR. GROVE: Well, I have a few questions. Since this symposium was arranged, I've been reading up on the literature.

I'd like to ask Dr. Moore if he has any information on the tonsils as glands concerned with growth, the hemopoietic system and blood production. You know these functions have been considered for many years. Dr. Moore, do you have any ideas on these subjects?

DR. MOORE: No more than in the general reticulo-endothelial system. I think that if there is anything to indicate that this is concentrated in the tonsils and adenoids—I missed it. I admit, of course, they do take part in the general overall reticulo-epithelial functions.

DR. GROVE: It used to be thought that the removal of large tonsils affects growth. The old textbooks bring that out emphatically, and doctors in those days were very much concerned.

DR. MOORE: Dr. Joyner, there's one thing I would like to mention at this point. We've given an indication that the tonsils are very important as far as immunity is concerned, up to at least the age of 4 years. I'm very much interested in hearing and ear infections. I believe that, when the adenoids, even at the early age of six months, are severely hypertrophied, infected, and blocking the eustachian tubes, and you have repeated middle-ear infections, the adenoids can be sacrificed without any apparent interference with your immunity program; but, under such circumstances, we prefer to leave the tonsils in.

DR. JOYNER: Now, Dr. Grove, we talked about the nature of tonsils and adenoids, would you tell us a little bit about the normal

life history. In other words, we know that in children, at certain ages the tonsils and adenoids are normally larger during a sort of lymphoid growth period. I think it would be rather important to stress that size in itself, particularly of the tonsils, doesn't mean too much at certain ages.

DR. GROVE: I think when Dr. Joyner spoke to me last week about this subject of the development and recession or involution of the tonsils and adenoids, he had in mind the so-called normal individual. Of course today we know when you see children in the office it's rather hard to decide whether you have a perfectly normal child. We are basing this opinion on the past history. Doctors in general, (we won't be specific about the nose and throat physician and the pediatrician) have the idea that at birth and up to maybe two to two and a half years of age the tonsils and adenoids are small. They begin to enlarge or hypertrophy thereafter (we're talking of the non-infective type, of course) then before or around puberty, we have the idea that they go into involution. Now, that is the general idea of a normal course as far as the life history of the tonsils is concerned.

Dr. Joyner said he thought he'd have some present experts discuss this question of the tonsils and adenoids and I thought I'd go back to some of the past experts of the textbook group and see what I could find about how they felt. Well, much to my surprise, in the four leading textbooks of nose and throat diseases I could find nothing. Everybody was concerned with infections and complications. I could not find any discussion like we used to have years ago as to what was a normal course of the development of tonsils. In fact, the textbooks on nose and throat did not even go into any discussion of the normal tonsils so to speak.

Lederer, in his textbook in one little paragraph said, "the tonsils are small in childhood and hypertrophy in puberty." That seemed to be the general idea.

Then I looked in the pediatric textbooks (they're pretty good on this I think). Most of these textbooks are in the Pediatric Library and I was in the Main Hospital Library where there were about three pediatric textbooks which I looked through. Holt and McIntosh were the first and I was very surprised that I could not get any general idea of what they felt about the subject. I know from my contact as a student of Dr. Howland's (Professor of Pediatrics at Johns Hopkins Medical School), how he felt about the normal tonsil and its course of development and what to expect and what,

you might say, not to expect pathologically. Then I decided I'd look further and fortunately Dr. Joyner has the late Dr. Abt's textbook in the Library. I thought it looked pretty worn and looked at the date. It was 1923. Well, he had more in that book than six or eight of the nose and throat and pediatric books that I looked over, so I said, "I'll see what he had to say" and it shows that back in those years there was a lot of discrepancy about this subject of the life history of the development of the tonsils and adenoids. Of course, all those men I suppose were very good language students and most of his references were in foreign languages, particularly in German, so some of his quotations naturally were from the German.

He said just what Dr. Moore said about the formation of the germinal centers. He wrote of the adenoids first, stating that about the first year of life they were small and reached a maximum about the sixth year. He felt in the normal patients at that age the adenoids would occupy probably half of the nasopharynx. We know that at times they occupy a lot more but he thought a general description would be about half of the nasopharynx and then at about six or seven years of age, they begin to involute, or, in other words, atrophy and about puberty this was complete.

He then makes the statement that according to most of the investigators there was a lot of doubt about the involution. They thought most of the adenoids would go on to maturity before they involuted and we agree on that. I think he was making it a little bit conservative and I got the impression they were not decided just when to draw the line on the adenoid as having finished its development and when it was going to atrophy. We know, I'm sure Dr. Moore does, from clinical experience, that the adenoids may stay large longer; some of the largest adenoids are found in the late teens and the twenties. This is beyond our discussion here, but we know that the adenoids don't always follow that pattern of involution.

Now getting to the tonsils, the discussion was very interesting. I think Dr. Moore would be interested in hearing this. He probably learned years ago as I did, and maybe like so many other things we have forgotten, Dr. Abt made a point that at birth and the first six months (I think the pediatricians probably know more about this age than we do, as they see more of the throats) the tonsils are more horizontal and they come over towards the midline. Then within the first year they begin to straighten out in the fossa or the tonsillar sinus and become more in the vertical plane and I agree

as I remember this development—I don't see too many children six months or under. He said by two or three years of life this development is complete, but I think this may be a little bit too long. In other words, the tonsils assume the position probably earlier but it is interesting that at birth they are in that horizontal position.

Regarding the changes that the tonsils undergo during childhood, he was very undecided. He said it was not clear to the men at that time. That is his version but, of course, a lot of the doctors in 1923, that isn't ancient I know, knew more. He quotes Genter who in turn was quoted by Gundobin, who examined the size of the tonsils. Talking about the atrophy, he found in the newborn the size of the tonsil was ¾ of a gram and from then on up to about five years, it increased rapidly. This is in line with the development or hypertrophy we are talking about. Then the weight went up to 1½ grams at about five years of age and he also quoted Hett and Butterfield as saying that atrophy began at about five years of age and continued on to puberty, but he said that there was still a lot of disagreement among the investigators about that involution or atrophy at that time and I think that is wrong.

In other words, clinically we see patients under two or two and a half years with small tonsils and then they begin to enlarge, to eight or ten years. Probably within that range they have increased to their maximum and then they begin this change or atrophy.

I certainly do not think they come to puberty at this change or atrophy. We know it is starting but we also know it runs beyond that. Dr. Moore was getting into a little clinical discussion and I wasn't going to make too much of an issue as we were talking about the life history of the tonsils. If you look at the throat you can't tell if a lot of these patients have had trouble, infections in other words, so instead of seeing a small tonsil at one, one and a half or two years of age, you may see a hypertrophied tonsil—and that is often, of course, from infection. On the other hand, when you get up to eight or nine years, a lot of those tonsils are already atrophied and certainly beyond that up to puberty in some patients. So you cannot always look at throats and say by the size whether or not a tonsil is infected. We do know that hypertrophied tonsils—and I think Dr. Moore mentioned that—are not always infected tonsils by any means.

Dr. Moore spoke of immunity. I think a lot of our hypertrophied tonsils in children are more immunologic, as far as the patient is concerned, than some of the smaller ones. I think it would do more

good to leave those in unless there is a definite reason to remove them. When we come down to it, I think of our clinical picture as the life history of the tonsil. It must be based a lot on the history of the patient, not only locally, as far as symptoms in the nose and throat are concerned, but from a general standpoint as well.

DR. JOYNER: Dr. Levine, you've heard this discussion. The first thing I'd like to ask you is what (from looking into many mouths at many ages) would you say, without looking it up, has been your experience on this question of size? Because it seems to be of some importance; and I think we have advanced a great deal when most of our otolaryngologists immediately state that the tonsils' size alone is not definitely a sign they ought to come out. What has been your experience?

DR. LEVINE: During the early years of life or, I should say, the first year, the tonsillar tissue is very small. If you want to make a note in your records, you're likely to put down "tonsils not seen" or "small". In most children, under the age of one, you can hardly see the tonsils; then, after that age they start to get larger and larger, depending upon the individual child. I believe that most of us pediatricians today do not go too much by the *size* of the tonsils. Some children get along very well, and go into adult life with fairly large tonsils. But there are two particular types of large tonsils of which we ought to speak—these are first, tonsils that are so big that a child may have difficulty swallowing solid foods, and another type, where children are able to swallow solid food, but the tonsils are so large and the passages so very small, that danger exists in those children developing a severe pharyngitis, and that the tonsils will close up. I've seen two cases that almost went to tracheotomy because the large tonsils had become infected and had practically closed up the pharynx. The child hardly got air through the passage because the tonsils were so large!

Those really are the two situations where I think the size of a non-infected tonsil should be an indication for removal. I always worry about the tonsils that almost come together in the center, but if a parent doesn't want them taken out, then the thing to do, as soon as the child gets any kind of infection, is to start immediately on antibiotic therapy.

I was an intern in the early 20's, and at that time they were taking out practically all tonsils. There was a great deal of fear that rheumatic fever could be caused by tonsils. Then, in the late 20's, or in the very early 30's, we began to learn a little bit more

about the value of tonsils. I believe work was done up in Rochester, N. Y., if I'm not mistaken, with two groups of children. It was concluded, on the basis of this work, that when the tonsils were taken out, the children developed fewer sore throats but many more coughs, as if the tonsils had been the barrier (that Dr. Moore spoke about), and held infections in the upper respiratory tract.

Then, a number of other writers, including Dr. May Wilson, who ran the Rheumatic Fever Clinic over at the Nursery and Child's, and at New York Hospital, reported on the basis of hundreds of cases that she found no difference at all in those children who had their tonsils out, and those children whose tonsils remained, on their susceptability to rheumatic fever. So, after that we weren't so apt to take out tonsils; certainly, we hesitated to take them out before 2 years of age because of the tendency of the pharyngeal lymphatic tissue to return as if the body needed this tissue for some reason. I'm not sure we know today the whole story about the tonsils in the young child, but a good deal of lymphatic tissue does return if the tonsils are taken out too early.

DR. GROVE: I want to say in the first place as I said a while ago that I do not think a hypertrophied tonsil has to be removed if it is not infected. I mean in many patients they act as a barrier to infecion so the first thing that I would bring out, and it is discussed in most of the textbooks, would be to explain the question of size Dr. Levine has mentioned the size as being important in certain patients and an indication for tonsillectomy.

I don't know what Dr. Moore thinks, but I've seen only two or three children where I had to take out tonsils because of size. Now if you get a tonsillitis or quinsy, as Dr. Levine mentioned, it is necessary, but I'm talking about the hypertrophy in itself.

Many times I look at these children's throats and I wonder how that child can breathe but he does all right. If he gets an acute infection then we have an indication for operation but otherwise not.

It also interests me how these children, youngsters under two or three years of age, can swallow with these large, hypertrophied tonsils but I don't think I've seen over one or two who couldn't swallow. I believe Dr. Moore mentioned the same thing. They will ruminate, that's frequent, but the food goes down. So I think size occasionally is an indication for operation but I would put that last.

My first indication would be, naturally, recurrent or severe attacks of tonsillitis. I mean you see the patients and they have swollen tonsils, the old follicular tonsillitis, or a peritonsillar abscess or quinsy, as we used to call it, enlarged glands, fever and the general reactions to an infection.

Ear infections are a definite indication for operation and of course when we talk about tonsils in children at the age group that we are discussing we have to include the adenoids. If you want to take maybe children under six months, you can do an adenoidectomy but we are talking about older ones and if they have had repeated ear infections, as Dr. Moore has already emphasized, which have affected the hearing, operation is imperative.

I talked to a couple of confreres on our Staff (unfortunately they couldn't be here) and one doctor who is at Bellevue told me there was a surprising number of children brought to Dr. John Daley Clinic with deafness. They had had an infection, they had been put on antibiotics and they had developed deafness. They didn't have an otitis media with an abscess. If a general practitioner looked at the drum, I think he would say they had a serous type otitis media but we would call it, a secretory otitis.

They have been given antibiotics and passed along from episode to episode, hearing getting worse and he mentioned several he had seen down there in the last couple of days, 30 decibels of loss in hearing, conductive type, and nothing had been done over a period of months. When they see them, of course, their adenoids are hypertrophied as Dr. Moore described before, blocking the eustachian tubes and the tonsils are also infected. The drums were dull. In some you could see the fluid and in some not, but when you open the drum and drain the fluid out, within 25 hours their hearing goes from 5 to 30 decibels.

You have seen this type of infection and I have seen it, so we would have to say that ear infections whether acute otitis media with an abscess or without—and today with the use of antibiotics, we don't see so many abscesses—a change in hearing, is another indication for operation.

I said before, hypertrophied tonsils were not to me too much of an indication for operation unless there was definite disease but in some children with hypertrophy of the adenoids where the nose is blocked and they have had sinus trouble, there is an indication. In some of our allergic patients who have an allergic rhinitis and they are sensitive to allergens and they have had any number of injections with no result, the blockage of the adenoids will increase the secretions in the nose, coming, as Dr. Moore described, from the crypts of the adenoids and the secretion keeps the turbinates boggy. The patients are stuffy and sniffy. Of course it interferes with their breathing. This may clear up in a certain number of patients but I think in some of these patients the mechanical obstruction of the adenoids has to be considered as an indication for operation.

Next as far as the tonsils are concerned, I would say an additional indication for operation is the general toxic reaction from the tonsils. We know this is open to discussion—that is, the effect of the tonsils on the general system and Dr. Levine has already brought out what Dr. Kaiser reported from Rochester, New York in 1931. We thought the nose and throat specialty had had a stick of dynamite put under it and we might as well get out of practice. Dr. Kaiser was certainly an ardent worker and he made a lot of investigations. He was quoted by Dr. Levine as having investigated 2200 cases but I looked up the literature and it was really 4400 cases. When he published his investigation he brought out the fact, as Dr. Levine said, that certain of the systemic infections are not influenced by tonsillectomy, meaning especially the colds and I think most of us agree that we would not do a tonsillectomy on the basis of colds along with no complications in a child in good general health. Bronchitis and laryngitis, he brought out particularly, were not affected and if you want to be honest, some of the patients are worse following operation when there has been no indication to take out the tonsils that have hypertrophied.

Lymphoid tissue does hypertrophy on the pharynx (we see a lot of this in our allergic patients especially after operation). In some of the best tonsillectomies however, little nodules outside the capsule, or as Dr. Moore said, in the other glands, or along the base of the tongue begin to grow back if they are done in early life. So there is a reason why we don't want to do tonsillectomies too early in childhood without indication in these systemic cases. If you do, in some of them, of course, the results are bad.

There is a reason why we don't want to do tonsillectomies too early in childhood without indication in these systemic cases. If you do, in some of them, of course, the results are bad.

Now there has been a lot of discussion regarding rheumatic fever. I am not giving that as an indication for operation unless it is associated with acute hemolytic streptococcus tonsillitis. In certain cases where the pediatrician agrees that the tonsils have a deleterious effect on their general systemic condition such as nephritis, we have to consider operation. So I think where there's a general systemic disease and the pediatrician agrees there is indication for operation, it should be done. However, I am not in favor of promiscuous tonsillectomies. I think if the indications are followed carefully and the operation is done properly, the results will justify themselves, but I agree with what you have all said that we have got to use good judgment.

DR. JOYNER: I agree with what you've said, but I would like to emphasize one of the points. Dr. Grove said "infections of the tonsils". As I understood it, you had an "s" on the end of that infection, and you meant not just one infection, but repeated?

DR. GROVE: Repeated infections.

DR. JOYNER: And that does not mean, in your eyes, repeated upper respiratory?

DR. GROVE: I mentioned that.

DR. JOYNER: In other words, specific infection, of a specific organ which is repeated?

DR. GROVE: That's right.

DR. JOYNER: Dr. Moore, I see you've been taking notes, would you come in on this and give us your indications, if you can add any or subtract? I'd like to know what you make your decision on?

DR. MOORE: I agree completely with what Dr. Grove has said regarding the recurring effects of tonsillitis, peritonsillar abscess, and gland and ear infections. I would particularly like to re-emphasize hearing impairment. We are seeing this more and more. I would say that 20% of the children I see for a question of tonsillectomy, have a 10 decibel or greater hearing impairment when I see them the first time. Now, I suspect that this is not usual, because I think I am getting a select group of cases with this problem.

I would like to emphasize mouth breathing. As you know, from your nasal physiology, it is not inspiration that has to do with the development of the sinuses in the child, it is expiration. After the inspired air is warmed, on expiration, by the nature of the turbinates the configuration of the nose, air eddies and currents are set up; so that this expired air goes into every sinus cavity. It is this slight positive pressure on expiration that stimulates development of the sinuses. Thus, facial configuration is dependent upon this development.

Now, in these children who are allowed to go on indefinitely with the nasopharynx completely blocked with adenoids (tissue), you will see a typical adenoid facies with narrow nose, narrow arch and pre-maxilla, and, if you look at the palate, you'll see a high-arched palate.

Unless these adenoids are taken out, these children are good candidates for an orthodontist. I think that's very important. I also think we're seeing more peritonsillar infections in children as a result of recurring infections where they've been treated with antibiotics. I'd like some confirmation or denial of this. My

impression of the children I'm seeing now is that the tonsils are more difficult to remove than they were when I was a Resident, for the simple reason that the entire superior pole in at least 60 or 70% of these tonsils is completely imbedded. In other words, they've had an exudate outside of the tonsillar capsule, and there is a complete scarring and exudate. You can actually see it just as if they had had a true peritonsillar abscess, where the tissues are frozen, and that must be cut out with sharp dissection; you can almost tell that when you pick the tonsil up with your tenaculum.

There's another point I think we may overlook, and that is, that in these children who have had repeated attacks of acute tonsillitis, cultures have shown that at least 80% of these tonsils contain hemolytic streptococcus by tissue culture. That doesn't mean all these children should have tonsillectomies, but I think you should be aware of that, because this is a potential source of infection.

If this child's resistance is lowered by various factors and the child is carrying active infection in the tonsils, then they are very prone to develop an acute exacerbation of the infection.

DR. JOYNER: I think we pretty well agree on what you've said on indications. We'd have to separate a little bit the tonsils and the adenoids. At first we agreed that both tonsils and adenoids varied in their size; but if we take the adenoids alone—if they occupy roughly more than 50% of the nasopharynx, or they cause obstruction either to the eustachian tube or to the normal respiratory movements—definite harm results

As far as the tonsils go, we come down to one indication, which is chronic infection, and we discussed what chronic infection is. I would like to bring one point up, which while an old one, I think is perhaps a crux in chronic infection: Is this tonsil re-infecting itself, is it still serving its purpose more or less by blocking another germ?

Now, we assume, and I think you pediatricians will appreciate it, that if a child gets more tonsilar infection than other children in his group, and is exposed considerably less, with attacks every month or six weeks, then he has re-infected himself. That would be a true indication for doing a tonsillectomy. Everyone has stressed the importance of hearing, and I'd like to ask the otolaryngologists here a question which has always bothered me. Unfortunately my clinical experience has been that all these ear infections don't clear up when you take the adenoids out. A good many of them are

sent over for radium next year. I've been told that the reason for that is that in removing adenoids, you cannot scrape around the eustachian tube, and you leave a nice tuft of adenoids there; so while you remove the contiguous infection, you leave that one area. I just throw that in because I've been a little disappointed in the last two years in the number of repeated otitis cases I've had. Dr. Moore, would you like to say anything about that?

DR. MOORE: I'd like to clarify that, and I think I can. Now, as far as the adenoids are concerned, we do—at least *I* do—a meticulous removal of the adenoid tissue, and when I finish with the nasopharynx, the mirror shows that there is no adenoid tissue. I mean I check it with my fingers and the mirror. You will see that in the eustachian tube arc and by the appearance of the eustachian tube, there is a great variation in mass as regards the posterior cushion. That means that there is lymphoid tissue in that cushion, and lymphoid tissue and mucous glands that extend up approximately half the length of the eustachian tube.

If an operator, who is not well informed and is over-zealous, injures that part, he does more harm than good, because he can cause adhesions and this will cause hearing loss. Therefore, we remove the adenoid tissue; then we check the hearing in approximately six weeks. If the hearing hasn't come up in six weeks (and approximately 1/10th of them will not) by then—if there is evidence of eustachian tube obstruction—they should have the radium applicator. I believe that quite strongly, and I don't know any other satisfactory way of treating the eustachian tube orifice. I do not believe, in general, that x-ray therapy (giving the 100 R by 3-6 treatments, as is routine at varying intervals) is at all as successful as the monel-metal nasopharyngeal applicator. I think it's far more effective, and I've checked this repeatedly, using the nasopharyngeal pharyngoscope.

DR. JOYNER: One reason I brought it up is that I think some of us in pediatrics get a little disappointed when, after an adenoidectomy, quite a few continue to come in with repeated ear infections. However, an adenoidectomy shouldn't be discarded simply because 10-20% get repeated infections.

DR. MOORE: I didn't give a routine answer either, because there's another group—and it's a large group in here. I'm sure Dr. Grove was just about ready to mention it, and that is allergy.

DR. JOYNER: I thought we'd get to allergy a little bit later.

DR. GROVE: I agree 100% with Dr. Moore, but I would like to go a little bit farther in that discussion about lymphoid tissue in the

eustachian tube. You remember Polvogt, Crowe and Guild at Johns Hopkins made serial sections of the eustachian tube and proved that what Dr. Moore said is true. That is where Crowe got his basis for the nasopharyngeal radiation of the eustachian tube.

Dr. Moore mentioned radiation with x-ray as not being effective. I think there is only a group of pediatricians here and I do not see any radiologists, but I'm afraid such radiation is "out of the window". Most observers, based on reports, fear the development of cancer of the thyroid and nasopharynx or changes in the bone marrow. The other point I was going to emphasize about these repeated infections, as Dr. Moore said, are other factors: malnutrition, a patient in poor condition, receptive to infections, and especially the allergies—the treatment of which is often neglected, but which to me is very important.

DR. LEVINE: I just want to ask the nose and throat specialists a question: how often during the year or two following a clean adenoidectomy, do you get enough regrowth of lymphatic tissue to be responsible for some of these otitis medias?

DR. MOORE: I would say that it is extremely rare in the absence of severe allergy and repeated, uncontrolled, untreated infections.

DR. JOYNER: Would you say that is so in a child under 2, who had had adenoids out?

DR. MOORE: No, I think you're getting into—

DR. JOYNER: Those are rare cases.

DR. MOORE: I think with those, you assume you will probably have to do a second procedure later on. You have a different picture in those children, and if you check with the nasopharyngoscope, you'll find they have not re-formed true adenoid tissue with typical crypts and mucous glands; but they have a diffuse lymphoid hyperplasia and that presents a little different picture.

DR. GROVE: I'm sure that all of us here have seen patients in the clinics and coming into our offices with adenoidectomies and T & A's whose operations were not done as meticulously as Dr. Moore described. We know this is the ideal way and it is being written up every month or two in our nose and throat journals. Dr. Joyner made a point about adenoids growing back if operations are not done carefully.

I think lots of times the operation is done very hurriedly. Now, we might as well be frank. We know in some parts of the country, you pediatricians do adenoidectomies, T & A's, and general

surgeons do them in a lot of places now. I have watched them. They scoop out some adenoids. In other words, lots of times, it is poor technique. We have to agree on that. Isn't that right, Dr. Moore?

DR. MOORE: Good results can only be obtained by a good adenoidectomy and tonsillectomy using the dissection and snare method and good hemostasis.

DR. JOYNER: Just one other point before we go to allergy, Dr. Levine?

DR. LEVINE: On removal of adenoids that are so large that a child is a mouth-breather, you often find many of these children are thin children with poor appetites. They can't smell, so that much of their food is tasteless. Once the adenoids are removed, these children immediately pick up. The food becomes more palatable once the sense of smell is restored, and they eat very much better.

DR. JOYNER: I'd like to deviate a little bit from the absolute indications that we've been talking about, and ask Dr. Levine to say a little bit about the psychological aspects. Now, as pediatricians, we're supposed to be and usually are rather interested in the psychological aspects of T & A's, and certainly, the way the tonsils and adenoids operations used to be done, you couldn't have thought up a more cruel experience to put a child through. I'd like Dr. Levine just to mention some of the psychological aspects of how we can help these children.

DR. LEVINE: Again, it depends on the age of the child. Most children under the age of 5, unless their hospitalization can be carried out correctly, from a psychological point of view, can be hurt a great deal. I think you all know the great trauma suffered by the young child—separated from the mother, brought up to an operating-room, seeing masked figures walking around, experiencing a mask pressed on the face, waking up with an intensely sore throat, nauseated, and not finding one person the child seeks there—the mother. Because these children under five or six years of age are so completely dependent upon their parents, they can suffer a great deal of psychological damage. This has been demonstrated in a good many reports from various parts of the country.

I think most nose and throat men are fairly cognizant of this fact today and in many of the hospitals, they try to make the situation as attractive as they can psychologically. For one thing, they're likely to give the child some sedation beforehand, so that he has no recollection of leaving his mother.

In some hospitals, like New York Hospital, the children get rectal anaesthesia. I don't think that is advisable generally, unless there are skilled anaesthetists. I know in some of the hospitals they're afraid to use rectal anaesthesia, but if that can be done, the child usually has no recollection of the experience at all. But the child should know something about the operation ahead of time. There are a number of good books out now. One came from the Children's Hospital in Boston. Another book is called "A Visit to the Hospital". This relates much of the experience, so the child knows: 1—it's not going to hurt him; 2—how much better he's going to be after it. Unfortunately, there are still parents who lie to their children. Recently, I saw a little girl at one of the hospitals walk in with a basket in her hand, thinking she was going to a picnic!

Another real advance today is that a good many of the nose and throat specialists are permitting the mother to remain with the small child so that when he wakes up, the mother will be there; and they also arrange for the mother to stay overnight in the child's room.

In older children, I don't think we have to go to these extremes psychologically, because once a child gets to the age of six, he has been going to school and has learned to accept a "mother substitute". But before that age, there's nobody that can take the place of the mother, not even the kindest nurse.

If a child also is going through a bad psychological period and is emotionally upset at the time a tonsillectomy and adenoidectomy is advised, it's undoubtedly better to wait for a while until the child has improved emotionally. If he's stuttering, stammering or having other signs of emotional difficulty for one reason or another, the operation can be delayed for a while. But generally speaking, I think many of the nose and throat men today are fairly cognizant of psychological conditions relating to children. They don't rush them; they try not to upset them or hurt them; they make every effort to be friendly with the child and to get his confidence, and if they possibly can, they let the mother fit into the picture.

DR. JOYNER: I know from experience that most of the otolaryngologists we have here today feel pretty much the same way about it, but I would ask Dr. Moore if he would say something just very briefly about anaesthesia—what does he consider safe, the best type of anaesthesia for the operative procedure and for the psychological aspect?

DR. MOORE: I think I can summarize this very briefly, but, before doing that, I would just like to make one plea—that would be to try to avoid the psychological insult to the mother by doing just this. I think the otolaryngologists and the pediatricians should know what hospital accommodations are available, and they both should give the same advice to the mother. We know that a child can be taken care of beautifully in a private room, where the mother spends the night of the operation with the child; we know that they do nicely with other children who are supervised, and the mother can be with them for a reasonable period. But it is a terrible problem when the mother is referred for the tonsillectomy and told that the child must have a private room, when the chances of getting a private room are one in a hundred. Then, you have a psychological problem that is almost insurmountable; whereas, if the situation were faced from a practical standpoint, the otolaryngologist and the pediatrician would rarely have a problem.

Now, regarding anaesthesia, I think rectal pentothal anaesthesia is ideal, and I have seen no trouble at all with it, but I think a well-run recovery-room is essential because for a period of 1 to 3 hours, these children are not able to look after themselves. They have to have a nurse in constant attendance.

Now, if under these circumstances, a child were given pentothal and then sent back to a single room where the nurses were up at the end of the corridor and that child was left to himself (even though there were sideboards on the bed), I think it would be a very dangerous thing. Not only could the tongue fall back and block the breathing, or the child's breathing become blocked by bed-clothes or other conceivable accidents, even to the point of the child's getting up, falling out of bed, and not knowing what he was doing. Under the proper conditions, I believe rectal pentothal or avertin is the ideal way to take care of these children.

We have also had the experience with our private children, whereby they have been given sedation before going up to the operating room. From a practical and psychological standpoint, by survey, this has worked almost as well. Would you say that, Dr. Levine?

DR. LEVINE: Yes, I fully agree. They don't remember leaving their room, or anything until they awaken.

DR. JOYNER: Dr. Grove, do you agree?

DR. GROVE: I agree with Dr. Moore entirely. We've used avertin, as Dr. Levine said. I've used it for 30 years. I was in the Johns Hopkins Hospital when it was first used, in 1928 or 1929, and it

works perfectly well if you have an anaesthetist who knows how to use it, but as he said, a lot of nurse anaesthetists come here and they have never heard of rectal anaesthesia. I understand the Children's Hospital in Boston has gone back recently to avertin instead of rectal pentothal, but his points are well taken.

DR. MOORE: I would like to add one thing about rectal pentothal; it is not well recognized. These children are given rectal pentothal after an enema. They have an enema the night before, and they're given rectal pentothal by weight. Let us say a six-year old child, perhaps will be given 6 or 7 cc.—I'm speaking "roughly" now. If that amount of rectal pentothal is allowed to remain in situ during the procedure, you're very apt to get depression during the operation and that can be serious. We attempt in every case to remove as much of that pentothal the minute the child arrives on the operating-room floor, not *in* the operating-room, but outside, and we recover of that 6 cc. on the average 3-5 cc. That means only 1-3 cc. has been absorbed. From my experience, this is the most practical safeguard.

VOICE: Why don't you give 1 cc?

DR. MOORE: Apparently 1 cc won't suffice, because the absorption is on the surface areas and a certain amount of surface area must be covered; but you can get it back. 3 to 5 cc. are wasted, but maybe it wasn't wasted.

DR. GROVE: May I ask Dr. Moore one question that's of interest to me. Here we use the procedure very little. Would you comment on intubation of children for T & A under 10 years of age?

DR. MOORE: I have little experience with it, because our anesthetists do not routinely intubate children under age 6. One of the reasons for this is that in children of this age it is very much in the way in a child's throat, with enlarged tonsils, and in the second place, we all know that endotrachael anaesthesia by the oral route is much more apt to cause trauma to the larynx. Because of the curve, the larynx is forced back and they're very apt to develop granulomas over the vocal processes. Incidentally, we do not give rectal pentothal after 9 years of age.

DR. GROVE: I'm glad you said that. I feel the same way.

DR. JOYNER: You apparently agree and we won't have further discussion on that.

Now, I'd like to cover one other thing before we throw the floor open. I think this question of children with infectious asthma is a special consideration. Dr. Grove has had a great deal of experience with it. It's a moot question, but I'd just like him to

discuss whether he feels that that is any added indication for the already stated indications for doing tonsils; in other words, would you take them out quicker in a child with infectious asthma?

DR. GROVE: I think I would say I might take them out a little more quickly but not necessarily so. We try to stick to the indications of infection present in the tonsils, and if you're dealing naturally with a patient who has the infective type of asthma, it is different from the other patients with the skin sensitive type, foods, or inhalants.

Dr. Moore emphasized occasionally there are organisms in the tonsils which we don't get on the surface and it is true. We do a lot of tissue cultures and we may sometimes stretch a point a little to get the tonsils for culture, but by and large, I'd say that we don't do it promiscuously and I would like to emphasize here if a child is very allergic with a lot of skin sensitizations that you can demonstrate, do not take the tonsils out until after you have given the child the benefit of medico-allergic treatment. That has been my experience and then if they do not do well, you can operate.

I think a lot of these children will get better and their adenoids may shrink; they may not be 100% well but if they are not obstructed completely give them the advantage of medical treatment first. If their condition is such that they are not losing ground and you feel that those other factors are more important—the danders or the inhalants, and in certain children, you run into foods—try the allergic treatment first and later you can always take out their tonsils and adenoids if necessary.

DR. JOYNER: Do you agree with that, Dr. Levine?

DR. LEVINE: I think many of us pediatricians hesitate to take out tonsils in children who are highly allergic but who have not asthma, in the fear that we may later develop bronchial asthma following a tonsillectomy. Do you feel that way, Dr. Joyner?

DR. JOYNER: I haven't looked too much at it. On the other hand, I certainly have come to the conclusion that, as Dr. Grove said, I don't believe there's any difference in indication whether they've got asthma, or rheumatic fever, or anything else. There are certain specific indications which we've gone over which are chronic infection in tissue in the body, and if an individual has infectious asthma, and he has chronic infection in his tonsils, I believe he's going to get more asthma. If he doesn't have a chronic infection in his tonsils, you'll probably be doing him harm by removing a barrier which may help him.

DR. GROVE: Dr. Levine opened up a discussion here—a question about removing tonsils in the allergic child who is a pre-asthmatic. Allergists are working on that problem. A paper was presented at the meeting of the American Academy of Allergists last October in Chicago, by a group in Brooklyn. It's going to be published. I won't discuss it. We don't feel too strongly about it, but you'll see it published by Dr. Markow & Associates. It's a question that ought to be discussed some time at one of our meetings as it is very important. I'm not in agreement.

DR. JOYNER: I think if there's any question from the floor, we'd be delighted to have it.

RESIDENT: With regard to the psychological aspect of the problem of having the mother of the child for T & A sleep during the first night, in our hospital, most of—perhaps more than 70% —of the T & A cases are ward cases, and perhaps only 30% or even less are private cases. What would be your suggestion for the children in the wards for T & A? Should these children have their mothers in the ward? From experience here, I have noticed that ward cases after T & A are doing much better than the private cases, especially when these private cases have their Mother sleeping with them, or even a private nurse.

DR. JOYNER: Would you answer that, Dr. Levine?

DR. LEVINE: Did I understand you to say that the children whose mothers, or somebody they knew and could depend on stayed with them, did better than the others?

RESIDENT: No, the ward cases seemed to do much better.

DR. LEVINE: Not psychologically? I can tell you this very definitely. You cannot judge in the hospital what's going to happen to the child after he gets home; the effect this experience is going to have on the child is not going to show immediately. Many of these children, when they get home, have shown signs of an anxiety neurosis, even though in the hospital they might have been interested in the other children around, and tried to be calm—almost afraid to cry. These youngsters have been through a much greater, much more traumatic experience than those who haven't had their mothers. I might add that there have been quite a few papers published on this.

Some thirty-odd years ago, when I was an intern, we refused to let parents visit their children during the week—we let them visit only for one hour or two hours on a Sunday afternoon. The reason given was this: the nurses said the children would cry and cry,

and this could only be harmful to the children. Then, in the course of time, it was learned that it was much better for the children, even if they did cry every day, to have their parents visit each day even if the parting was an upsetting experience, the psychological trauma in not having the parents there at all was much greater. So today, in many of the hospitals, the parents are permitted to visit several hours daily. I might mention that in England, a few years ago, Parliament passed a law that mothers of children under the age of 5, could stay in the hospital with their children; they could live there, with all their meals, board, and everything free.

DR. JOYNER: I'd like to stress again that you cannot judge by the immediate behavior, what the later psychological effect will be. Some of the ones that are going to get the worst psychological trauma later on, will be the ones that will be the nicest and most quiet while they are here. On the other hand, if you follow these children, as we do in the office, you'd be surprised at the number that start wetting the bed, who hadn't been wetting the bed before, have nightmares, who'd never had nightmares before, etc. I think that many of you who had T & A's done in the old beknighted eras, if you ask yourselves what childhood experience you remember most, a great many will say having their tonsils and adenoids out! It's something that sticks to them, but I think the crux of the answer, as you said, has two parts to it: one is what you mentioned, that you don't see the psychological aspects immediately; the second, a very practical one, is the cultural differences you see at times.

We have the same experiences occasionally with some of the children we have up in the wards here, where they cry not because they're going to be in the hospital, but because they're going home. We know that. But I think the crux of it is that this psychological harm we were talking about is not immediate. It's deeper than that, and that'ts the worst of it.

RESIDENT: I accept that, but yet, I still believe when we go through our late rounds about 10 or 11—sometimes even later—we see the ward cases sleeping like little birds. On the other hand, the private cases are restless. We have to force the private cases to drink liquids; on the other hand the ward cases take their glass of water or some other liquid with a straw, and they drink by themselves. They don't have to be forced.

DR. MOORE: Dr. Joyner, I was very much interested in this, because I think the doctor has a good observation, but I think it's part

of the story. They need *both* really. They need their mothers and they need other children too. It is my own personal observation that the ones who do best are the ones who have their rectal pentothal and sedation. The mothers come in, having told the child just what the program will be, and having told the child that she will be there. It is important that the mother be with the child at the time the rectal pentothal is given. In this way, the child remembers little if anything, until he sees the mother on coming down from the recovery room. The children who do best are not the ones who go into the private room and come down to find their mother alone in a strange hospital room; but the ones who go into the four-bed room where there are other children who have had or are having the same procedure. They have fellow-sufferers so to speak. From that standpoint, I think they do ideally. I personally believe they do best under these conditions.

DR. JOYNER: I would agree with both, but I would make one provision: I don't think it's the best psychological thing for one of them to be waiting to go up at 3 o'clock, and have the 1 o'clock one come down, vomiting blood all over, yelling, and thinking his throat's cut. Otherwise, I think you're perfectly right on that.

VOICE: I would like to ask if the great value of the recovery-room should be qualified; the way it's run today, it prevents a mother from being present. How can that be solved?

DR. MOORE: That's not a problem.

VOICE: How would you solve it? Have the mother come to the recovery-room?

DR. MOORE: No, the child doesn't remember when he is in the recovery-room and the mother should not go to the recovery-room. It would defeat the whole thing.

DR. GROVE: I know the cases our Resident is talking about. We get certain types of patients in here, and I'd agree with him very much—a lot of the patients are very difficult to handle but that is often due to the individual patient and the mother. Take 100 or 1,000 patients, not an isolated few. He's talking about that immediate first 24 or 48 hours. We're talking about their behavior later.

DR. JOYNER: The fundamental thing is not how they immediately behave in the hospital, but how they behave six weeks, six months, six years later. That is what we are interested in.

Are there any other questions from the floor? If not, I want to thank the panel very much for a most interesting and a most helpful discussion.

sulfa therapy suited to young tastes and tempers...

Employs the N' acetyl form of KYNEX to impart high palatability yet retain single-daily-dose effectiveness and rapid, high sustained action against sulfa-susceptible infections. **Dosage:** first day, 1 tsp. (250 mg.) for each 20 lbs.; thereafter, ½ tsp. daily for each 20 lbs. For 80 lbs., use adult dosage of 4 tsp. (1.0 Gm.) initially; and 2 tsp. (0.5 Gm.) thereafter. Taken once a day—preferably after a meal. **Supplied:** Each tsp. (5 cc.) contains 250 mg. sulfamethoxypyridazine activity. Bottles of 4 and 16 fl. oz.

CHERRY LIQUID AND 1-DOSE DAILY

KYNEX®

N' Acetyl Sulfamethoxypyridazine

ACETYL PEDIATRIC SUSPENSION

Lederle LEDERLE LABORATORIES, a Division of AMERICAN CYANAMID COMPANY, Pearl River, New York

a new infant formula nearer to mother's milk in nutritional breadth and balance

Infant formula

NEARER . . . in caloric distribution of protein, fat and carbohydrate

NEARER . . . in vitamin pattern (vitamin D added in accordance with NRC recommendations)

NEARER . . . in osmolar load

ENFAMIL IS ALMOST IDENTICAL to mother's milk in

- ratio of unsaturated to saturated fatty acids
- absence of measurable curd tension . . . enhances digestibility

*Trademark

Mead Johnson

Symbol of service in medicine